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Official Organ of
The American Phytopathological Society

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Official Organ of The American Phytopathological Society

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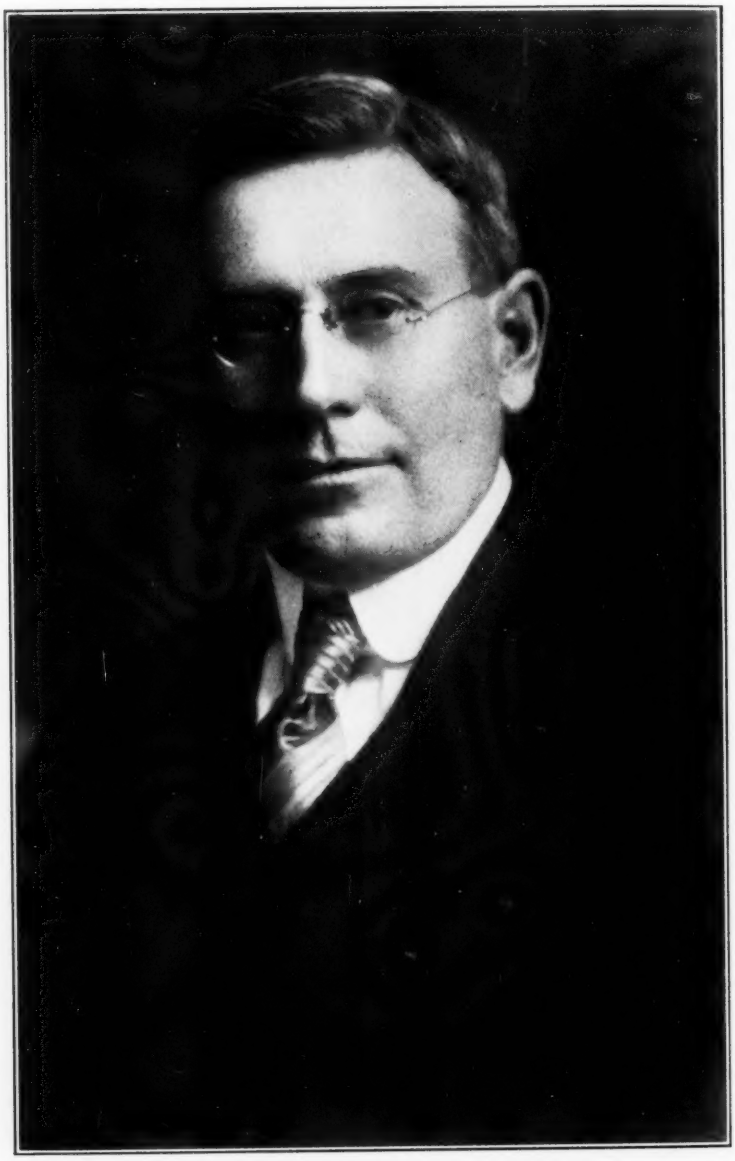

FREDERICK JOHN PRITCHARD

PHYTOPATHOLOGY

VOLUME 22 MARCH, 1932 NUMBER 3

FREDERICK JOHN PRITCHARD
1874–1931

WILLIAM S. PORTE[1]

The life of Frederick J. Pritchard was full of energetic and enthusiastic activity until the very hour of his demise. Endowed with a keen intellect and a powerful physique, verily he was a human dynamo and his interests in human affairs were legion. During the more than 12 years of close association with him, the writer never saw him relax but momentarily. To the incessant urge within him that overtaxed his heart, his friends attribute his untimely death, which occurred while seated at his desk in the Bureau of Plant Industry in Washington, D. C., January 13, 1931. At the time of his death, Mr. Pritchard was Senior Physiologist, Division of Horticultural Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture.

Born in Camanche, Iowa, on December 24, 1874, of sturdy Scotch-Irish stock, Fred Pritchard spent his boyhood years on a farm northeast of Council Bluffs, Iowa, where he had an excellent opportunity not only to develop into robust manhood but also to study at first-hand the mysteries of life as nature unfolded them. His analytical mind early pried into the secrets of wild life about him. In his mature boyhood he learned the fundamentals of zoology through the help of his father, who was a very successful veterinary surgeon. During all this time his noble mother was quietly instilling in her son high ideals and worthy ambitions, which became so ingrafted into his very nature that throughout his life Fred Pritchard never lost sight of those precepts and frequently referred to them. He attended grammar school at Persia, Iowa, walking 6 miles each day.

In early manhood he obtained a thorough knowledge of farming operations and the growing of farm crops by actual experience on the farm. As soon as he had mastered practical farming his thoughts turned to enlarged fields of learning. With money he had earned farming, he attended the Woodbine Normal School, Woodbine, Iowa, and as he advanced in his school work he took up teaching to earn his way through school. He then went to Omaha, Nebraska, and earned his way through business college, working

[1] The writer gratefully acknowledges his indebtedness to Dr. H. A. Edson, Chief Examiner, Civil Service Commission, to Dr. A. G. Johnson, Principal Pathologist, Bureau of Plant Industry, and to Dr. W. T. Pritchard of North Platte, Nebraska, for valuable suggestions and criticisms made in the preparation of this sketch.

evenings and vacations as clerk in a freight depot. With an alert mind and a sturdy body as his resources, he matriculated at the University of Nebraska in 1900, and majored in botany, plant physiology, and plant pathology, graduating 4 years later with the high esteem of the faculty. After graduation he continued for a short period as a graduate student at his alma mater and also served as assistant in botany. After this he accepted the position of instructor in botany at the North Dakota Agricultural College, later becoming assistant professor of botany, which position he held until 1907. There he became deeply enthused over the possibilities of breeding varieties of crop plants that would resist diseases. He resigned his position at the North Dakota Agricultural College to pursue graduate work in plant breeding and plant physiology at Cornell University. After 2 years he transferred to the University of Wisconsin, where he completed the residence requirements for the degree of Doctor of Philosophy in 1910.

It was while at Wisconsin that Mr. Pritchard, as Agent in the United States Department of Agriculture, became interested in breeding sugar beets. In 1911 he was appointed Assistant Physiologist and took charge of the sugar-beet-breeding work in the Bureau of Plant Industry. Extensive studies had been undertaken and much progress made in developing strains of sugar beets that yielded high percentages of sugar when it became necessary to terminate this work in 1914. Only those familiar with plant breeding can appreciate the immense amount of painstaking work involved in carrying out the preliminary phases of such a piece of research and the disappointment of the breeder when forced to abandon it before its completion. However, with undaunted courage, Mr. Pritchard embarked on a new field of endeavor, namely, the breeding of disease-resistant tomato varieties, in which he later attained the crowning success of his career.

Although the funds available were very meager, with characteristic enthusiasm and foresight he planned the work on such a large scale that some tangible results were obtained in a relatively brief time. He also laid the groundwork for a long-time tomato-breeding program. He quickly assembled varieties and strains of tomatoes from all parts of the world and tested them for resistance to the Fusarium wilt disease that was causing heavy losses in many important tomato-producing centers. Fortune surely smiled on this work, for within 4 years Mr. Pritchard presented to the tomato industry the Norton, Columbia, and Marvel varieties, which were wilt-resistant selections made, respectively, from the Stone, Greater Baltimore, and Marvel of the Market varieties. In the meantime, Mr. Pritchard had been selecting the most promising individual plants from a number of tomato varieties and crossing them. It was from this material that the Marglobe variety finally was developed. Probably no other tomato variety

ever has received a more spontaneous and universal reception by the industry than this variety.

Back in 1918, Mr. Pritchard was testing his wilt-resistant tomato hybrids for resistance to tomato blights of various kinds. In carrying out this phase of the work he showed unusual foresight. In 1924 the Department received an emergency call for assistance in saving the winter-tomato industry from the ravages of the nailhead-rust tomato fruit spot. The resistance of the Marglobe variety to this disease was so marked that the commercial damage from it proved negligible. It quickly became not only one of the leading shipping tomatoes but also the principal canning variety in many of the large canning centers.

Besides originating several other valuable tomato varieties, Mr. Pritchard made important contributions to the subject of plant-disease control by field sanitation methods and also introduced new copper combinations as fungicides. He contributed to our knowledge of the effect of soil fertility and cultural conditions upon the development of certain plant diseases and also worked out the life cycles of several fungi that are parasitic on tomato plants. In addition, he started work on breeding disease-resistant cucumbers and eggplants.

Mr. Pritchard possessed an unusual ability for making friends quickly. He had a personal magnetism that appealed to those whom he met, and he immediately won their respect and confidence. Managers of large agricultural enterprises who knew him eagerly sought his advice and counsel, not only on the subjects in which he specialized but also regarding matters in unrelated fields. Their confidence in him seemed to be implicit. On several occasions captains of industry who had met him have spoken to the writer regarding Mr. Pritchard's pleasing personality, his modesty, and his great store of accurate information on a wide range of subjects. He was very widely and favorably known by practical agriculturists all over the United States, and he also had much correspondence with agriculturists in foreign countries.

Only a person with extraordinary energy could have maintained such an active interest in so wide a range of subjects as did Mr. Pritchard. Repeatedly he would maintain intensive concentration on a pathological or breeding problem for weeks at a time and at the same time have some theoretical problem in an unrelated scientific field as his chief diversion, or hobby.

Fred Pritchard was a man of dauntless courage and endurance and did not hesitate to assume a solitary position on any subject when he considered his contemporaries were championing error. When he embraced an idea he never gave it up unless he had convinced himself of its fallacy, even though he stood alone against the field. Through the mellowing years he

evolved a philosophy of life that was at once optimistic and hopeful. Although he was an indefatigable worker and allowed himself practically no relaxation, he contended that mankind should have more time for leisure in order better to enjoy the beauties of nature and life; also that intolerance of the views held by others is engendered by the slavish grind that we impose on ourselves, which, in turn, causes much of the misery and irritation of our modern civilization.

Despite Fred Pritchard's penchant for ceaseless work, he was devoted to his home and family and spent every minute he could at home. This is the reason why he was rarely seen at public meetings except when duty called him. The loss of his young son Robert in 1926 was a shock from which he never fully recovered. However, he never complained and he bore his grief in silence. He is survived by his wife, Selma I. Pritchard, and only daughter, Dorothy.

Mr. Pritchard was a member of the American Association for the Advancement of Science; the Washington Academy of Sciences; the American Genetics Association; The American Phytopathological Society; the Botanical Society of Washington; Alpha Zeta, and Sigma Xi.

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BIBLIOGRAPHY

Internal infection of the wheat grain by rust. Science, n.s., **22**: 343–344. 1905 (with H. L. Bolley).

Rust problems: facts, observations, and theories. N. Dak. Agr. Exp. Sta. Bul. 68. 1906 (with H. L. Bolley).

A preliminary report on the yearly origin and dissemination of Puccinia graminis. Bot. Gaz. **52**: 169–192. 1911.

The wintering of Puccinia graminis tritici E. & H. and the infection of wheat through the seed. Phytopath. **1**: 150–154. 1911.

Relation of adaptation to the improvement of sugar-beet varieties for American conditions. U. S. Dept. Agr. Bul. 260: 43–48. 1912.

The Bureau of Plant Industry. Alpha Zeta, Quar. **13**, No. 4. Mar. 1915 (with W. W. Burr).

Correlations between morphological characters and the saccharine content of sugar beets. Amer. Jour. Bot. **3**: 361–376. 1916.

Some recent investigations in sugar-beet breeding. Bot. Gaz. **62**: 425–465. 1916.

Change of sex in hemp. Jour. Hered. **7**: 325–329. 1916.

The use of checks and repeated plantings in varietal tests. Jour. Amer. Soc. Agron. **8**: 65–81. 1916.

Experiments in transplanting sugar beets. Jour. Amer. Soc. Agron. **8**: 106–110. 1916 (with L. E. Longley).

Control of tomato leaf-spot. U. S. Dept. Agr., Bur. Plant Indus., Cotton, Truck, and Forage Crop Dis. Inv. Circ. 4. 1918 (with W. B. Clark).

Effect of spraying on early ripening of tomato fruit. Phytopath. **9**: 289–291. 1919 (with W. B. Clark).

Effect of copper soap and Bordeaux soap spray mixtures on control of tomato leaf-spot. Phytopath. **9**: 554–564. 1919 (with W. B. Clark).

Collar-rot of tomato. Jour. Agr. Res. **21**: 179–184. 1921 (with W. S. Porte).

Relation of horse nettle (Solanum carolinense) to leaf-spot of tomato (Septoria lycopersici). Jour. Agr. Res. **21**: 501–506. 1921 (with W. S. Porte).

Use of copper dust as a fungicide. Phytopath. **11**: 229–235. 1921 (with W. S. Porte).

Effect of fertilizers and lime on control of tomato leaf-spot (Septoria lycopersici). Phytopath. **11**: 433–445. 1921 (with W. S. Porte).

Isaria rot of tomato fruits. Phytopath. **12**: 167–172. 1922 (with W. S. Porte).

Development of wilt-resistant tomatoes. U. S. Dept. Agr. Bul. 1015. 1922.

Watery-rot of tomato fruits. Jour. Agr. Res. **24**: 895–905. 1923 (with W. S. Porte).

Tomato diseases and how to overcome them. Canner **56**: 124–125. 1923.

The relation of temperature and humidity to tomato leaf-spot (Septoria lycopersici Speg.). Phytopath. **14**: 156–169. 1924 (with W. S. Porte).

Tomato wilt and its control. Mark. Grow. Jour. **35**: 198–200. 1924.

The control of tomato leaf-spot. U. S. Dept. Agr. Bul. 1288. 1924 (with W. S. Porte).

Tomato wilt and varietal resistance. Seed World **17**, No. 5: 7–9. 1925.

Control of tomato blights. Canning Trade **49**, No. 22: 50, 52, 54, 56, 60; No. 23: 18, 20, 22. 1927. Also in Rpt. Maryland Agr. Soc. **11** (1926): 368–379. 1927.

The Marglobe tomato. Proc. New Jersey State Hort. Soc. **52**: 51–62. 1927.

Tomato varieties developed for wilt resistance. U. S. Dept. Agr. Yearbook **1926**: 727–731. 1927.

Tomatoes resistant to nailhead rust now widely planted. U. S. Dept. Agr. Yearbook **1927**: 630–633. 1928.

Tomato-blight control effected by destroying remains of the crop. U. S. Dept. Agr. Yearbook **1928**: 583–585. 1929 (co-author with W. S. Porte).

The Break O'Day tomato. Manuscript. 1931 (with W. S. Porte).

A PRELIMINARY REPORT ON RESISTANCE TO CURLY TOP OF SUGAR BEET IN BEAN HYBRIDS AND VARIETIES

W. W. Mackie and Katherine Esau

Curly top of sugar beet (Fig. 1) has been known to attack beans since 1919, when Carsner (1) reported its appearance in Idaho. Since then Severin, according to Carsner (3), has found that a large number of varieties of beans are injured by curly top of sugar beet. He noted especially that the plowing under of sugar-beet fields caused the beet leaf hopper,

Fig. 1. Varieties of sugar beets under test for resistance to curly top. Center 4 rows, susceptible variety. Varieties to the right and left, resistant. Adjacent to the bean plots.

Eutettix tenellus (Baker), to migrate to fields of beans that later developed severe injury from curly top. Severin and Henderson (5) found that naturally infected varieties of beans included Small White, Cranberry, Kentucky Wonder, and Long Red Kidney, of the species *Phaseolus vulgaris*, and Henderson Bush or Baby Lima (*P. lunatus sieva*). In addition, he experimentally infected with curly top the following varieties: Bayo, Blue Pod, Lady Washington, Pink, Red Mexican, and Spotted Red Mexican, of the *P. vulgaris* group, Scarlet Runner (*P. multiflorus*), and Burpee's Bush, Fordhook, and Lewis Limas (*P. lunatus*). Severin observed that the Lima beans showed in field and greenhouse much less injury from curly top than common beans (*P. vulgaris*), Scarlet Runner, and Blackeyes (*Vigna sinensis*).

The California Pink bean, long noted for its hardiness and high productivity, in California, was believed by Carsner (1, 2) in his first observations to be nonsusceptible to curly top. Later, Severin (4, 5) was able to induce curly-top symptoms by artificial inoculation in the greenhouse with infective beet leaf hoppers. Many California Pink bean plants failed to show symptoms of the disease, indicating, he believed, that there are races of highly resistant or immune California Pink beans.

Several years ago the senior writer secured a cross between a strain of California Pink and the variety Robust, a white pea bean, resistant to mosaic. Hybrids from this cross in the 8th generation and fixed for color, maturity, vine habit, and the commonly desirable characters of field beans were tested for their resistance to curly top. To this list was added Hopi, Baby Limas, and Butter beans. The varieties were planted in very short rows, 30 in. apart, in order to make certain that an abundance of infective leaf hoppers would attack each vine. Nymphs instead of adults were used in order to confine the insects to the actual vine upon which they were placed (Fig. 2). The rearing of the infective beet leaf hoppers, placing

FIG. 2. Bean hybrids and varieties under test for resistance to curly top. Sugar-beet hybrids under similar tests to the right.

them on the bean vines, and the taking of careful notes and observations on the resistance of the beans to curly top were the work solely of the junior writer, who has done considerable research on curly top of sugar beet. The

TABLE 1.—*Resistance to curly top in bean hybrids and varieties, 1930; planted May 13, 1930—Davis, California; inoculated with infective nymphs May 22 and 24*

1930 Row No.	Variety	Identification No.	Seed color	Number of plants	Grades of resistance		Number of plants with symptoms July 23	Number of plants without symptoms July 23	Bearing plants *Per cent*	Resistance Class (1–5)
					July 1	July 23				
1	Calif. Pink	4427	P	19	2	2	0	19	100	1
2	Robust		W	14	4	3	6	1	43	3
3	R×P	5(32)27	W	16	4	3	3	4	37	3
4	"	7(32)27	W	22	4	4	13	3	36	4
5	"	13(32)27	W	20	5	5	8	1	10	5
6	"	14(32)27	W	21	5	5	9	3	19	5
7	"	1(32)29	W	22	5	5	11	11	50	5
8	"	2(17)29	P	25	5	5	7	1	20	5
9	"	3(17)29	P	21	1	1	0	21	100	1
10	"	4(17)29	W	16	5	5	3	2	13	5
11	"	5(17)29	W	21	2	1	0	21	71	1
12	"	6(17)29	W	21	5	4	3	3	19	4
13	"	7(17)29	W	25	5	3	5	6	32	3
14	"	8(17)29	W	18	5	3	4	6	33	3
15	"	9(17)29	W	13	4	4	5	4	62	4
16	"	10(17)29	W	20	4	3	3	3	20	3
17	"	12(17)29	W	22	5	5	5	2	9	5
18	"	13(17)29	W	26	5	5	5	1	8	5
19	"	14(17)29	W	25	5	5	2	3	20	5
20	"	15(17)29	W	18	4	2	6	6	44	2
21	"	16(17)29	W	13	4	2	2	6	54	2
22	Long Red Kidney		R	13	3	3	?	?	77	3
23	R×P	1(59)27	P	26	1	1	0	25	81	1
24	"	2(59)27	W	21	5	5	12	2	71	5
25	"	3(59)27	W	18	5	5	2	5	11	5
26	"	4(59)27	W	20	5	4	8	3	25	4
27	"	5(59)27	W	24	5	4	11	2	29	4
28	"	6(59)27	W	17	5	5	5	1	18	5
29	"	7(59)27	W	17	5	4	3	1	18	4
30	"	8(59)27	W	26	3	1	6	18	70	1
31	"	9(59)27	W	13	5	4	4	3	46	4
32	"	10(59)27	W	24	5	5	1	5	4	5

TABLE 1.—*(Continued)*

1930 Row No.	Variety	Identification No.	Seed color	Number of plants	Grades of resistance		Number of plants with symptoms July 23	Number of plants without symptoms July 23	Bearing plants *Per cent*	Resistance Class (1–5)
					July 1	July 23				
33	R × P	11(59)27	W	12	5	5	0	2	17	5
34	"	12(59)27	W	14	5	5	0	7	29	5
35	"	13(59)27	W	16	5	5	1	11	19	5
36	"	14(59)27	W	99	5	5	1	1	0	5
37	"	15(59)27	P	26	3	2	17	8	100	2
38	"	16(59)27	P	26	5	3	4	9	27	3
39	"	17(59)27	P	38	2	1	32	0	74	1
40	"	18(59)27	P	19	4	5	0	10	32	5
41	"	19(59)27	W	18	5	4	1	12	22	4
42	"	20(59)27	W	25	5	5	1	13	12	5
43	"	21(59)27	W	23	5	4	2	9	55	4
44	"	22(59)27	W	17	5	5	1	9	24	5
45	"	23(59)27	W	24	5	5	0	8	0	5
46	"	24(59)27	W	25	5	5	0	7	4	5
47	"	25(59)27	W	22	5	5	0	7	0	5
48	"	26(59)27	W	26	5	4	4	9	15	4
49	"	27(59)27	W	25	5	5	1	4	4	5
50	"	28(59)27	W							
51	"	29(59)27	W	23	5	5	1	7	17	5
52	"	30(59)27	W	24	5	5	1	9	83	5
53	"	31(59)27	W	16	5	5	3	7	44	5
54	"	32(59)27	W	33	4	4	4	11	15	4
55	"	33(59)27	W	22	5	5	2	4	18	5
56	"	34(59)27	W	27	5	4	12	4	74	4
57	"	35(59)27	W	21	2	2	20	0	81	2
58	"	36(59)27	W	26	5	5	0	6	0	5
59	"	37(59)27	P	22	5	5	3	7	18	5
60	"	38(59)27	W	10	4	4	?	?	60	4
61	"	41(59)27	P	17	2	2	16	?	71	2
62	"	42(59)27	W	19	5	5	0	9	21	5
63	"	43(59)27	W	20	4	4	1	9	15	4
64	"	44(59)27	W	14	5	5	2	6	36	5

TABLE 1.—(*Continued*)

1930 Row No.	Variety	Identification No.	Seed color	Number of plants	Grades of resistance		July 23 Number of plants with symptoms	Number of plants without symptoms July 23	Bearing plants *Per cent*	Resistance Class (1–5)
					July 1	July 23				
65	R × P	45(49)27	W	14	5	5	0	9	21	5
66	"	46(59)27	W	21	5	5	0	10	10	5
67	"	49(59)27	Pinto	20	5	5	1	9	15	5
68	"	50(59)27	"	20	3	2	3	11	40	3
69	"	51(59)27	"	20	1	1	18	0	100	1
70	"	52(59)27	"	22	3	3	2	9	18	3
71	"	53(59)27	"	20	4	4	2	4	10	4
72	"	29(59)27	P	19	5	4	3	5	26	4
73	Greer's Kidney	41(150)27	Red mot.	8	3	3	4	3	75	2
74	Small White	4546	W	51	3	3	4	37	39	4
75	Blue Pod	4545	W	24	2	3	6	12	46	3
76	Small White	4421	W	35	5	5	0	23	26	4
77	Lady Washington	4424	W	15	3	3	11	2	93	1
78	Cranberry	4427	R mot.	15	1	1	0	15	80	1
79	Calif. Pink	4120	P	20	1	1	20	0	90	1
80	Gt. Northern	4365	W	10	3	3	4	3	70	2
81	Idaho Butternut	4365	W	10	3	3	4	4	70	2
82	Chili Bayo	4416	B	21	4	3	4	8	19	5
83	Jap. Nagazura	4413	B stri.	16	5	5	2	0	0	5
84	" Otenashi	4412	W	17	3	3	5	8	53	3
85	Korean Kotenashi	4411a	W	21	5	4	4	6	19	5
86	China Red	4414	R	15	5	5	1	4	13	5
87	White Navy	4438	W	19	3	2	7	8	47	3
88	Lady Washington	4443	W	16	5	4	6	7	50	3
89	Isbel's Wh. Marrow	4482	W	8	5	5	1	0	13	5
90	Hopi	(64)27	W	16	2	2	?	?	57	3
91	Baby Lima		W	19	5	4	3	4	26	4
92	Hopi	30(64)27	W	7	3	3	?	?	57	3
93	"	22(64)27	W	12	4	4	?	?	83	2
94	"	25(64)27	W	14	4	3	?	?	43	3
95	"	40(64)27	W	13	4	4	?	?	62	3

results of the tests with 96 hybrids and varieties of beans are presented in table 1.

DISCUSSION OF RESULTS IN TABLE 1

Correlation of seed-coat color with resistance. Red seed-coat color in beans is supposed by many to be correlated with resistance to curly top, and white seed coat, with susceptibility. This observation is furthered by the very high resistance of the common pink bean and other red and pink varieties. The actual tests at Davis, during 1930, showed the California Pink free from curly-top injury under heavy infection, but several red and pink varieties, including Red Kidney and China Red, were destroyed by curly top. Brown Bayo was also destroyed, and mottled and striped colored beans, including the Pinto, Nagazura, and Striped Kidney, were similarly injured. The mottled Cranberry, however, was found to be highly resistant.

Among the Robust × Pink hybrids the correlation between susceptibility and the white color did not hold, for a number of white hybrids were found highly resistant to or free from curly top. Conversely, some of the pink hybrids were destroyed by curly top. A greater proportion of the pink hybrids were resistant to curly top than were the white hybrids.

Grades of susceptibility. Five grades indicating the severity of the curly-top attack were used: Grade 1 (Fig. 6) includes those beans that show little or no symptoms of curly top and no apparent reduction in yield. Grade 2 (Fig. 3) indicates a visible but very slight curly-top infection; grade 3 indicates susceptibility; grade 4 includes highly susceptible plants;

FIG. 3. Robust × Pink hybrid (white). Heterozygous susceptibility. Grades 5 to 1—left to right.

and grade 5 includes those plants killed or rendered entirely unproductive by the disease.

The effect of curly-top infection is greatest when the bean seedlings are entering the 3rd- and 4th-leaf stage. When the initial infection occurs later the injury is correspondingly less. Mature plants show but little injury when subject to first infection at this stage. Certain varieties possessing a degree of resistance show marked ability to recover after the first injuries following curly-top infection (Fig. 4). In some cases the older branches

FIG. 4. Robust × Pink hybrid (white). Very susceptible. Grade 4. No beans produced.

on such resistant plants show pronounced symptoms of curly top, whereas the later growth does not indicate the presence of the disease. These differences were noted in a change of grade when observations taken July 1 and July 23 were compared. The reverse, or increasing intensity of attack, was not found.

Curly top distinguished from mosaic. The virus diseases attacking beans in California, curly top and mosaic, are common and wide-spread. Both are carried by insects, *Eutettix tenellus* (Baker) and *Aphis rumicis*, respectively, but the latter is known to be carried in the seed, which has not been observed in the case of the former. These diseases may be distinguished in the field by certain characteristics. The bean mosaic, more particularly, affects the young leaves. They are reduced in size and are cupped downwards. When held to the light areas of irregular size, both lighter green and darker green than the normal green are seen. Cupped or raised areas of dark green may be seen on the upper surface, and the smaller veins on young leaves do not appear transparent, as they do in the case of curly top. The curly-top-infected leaves do not show any mottling but are darker in color than normal and develop a puckering and an out-

ward cupping of the young leaves (Fig. 4). Both diseases cause a stunting of the plants. The small veins appear plainly clear or transparent. In the Robust × Pink hybrids there is positive indication that a satisfactory variety resistant to both diseases can be secured, for the Robust parent (Fig. 5) is resistant to mosaic and the Pink parent to curly top (Fig. 6).

FIG. 5. Robust (white pea bean). Susceptible to curly top.

In certain heterozygous families of this cross all grades of resistance to both diseases appear.

Species resistant to curly top. Four species of beans were tested for curly top, including common beans, Butter beans, Small Lima or Baby Lima, and Blackeye cowpea. All species included susceptible varieties, but the common bean appeared to be most susceptible, although immune and highly resistant varieties were found. The Butter bean appeared moderately susceptible in the limited number of varieties under test. The Baby Lima did not show the extremes of susceptibility, but no highly resistant or immune varieties were discovered in the limited number tested. There is evidence for the belief that a wide range of resistance may be found in all species of commercial beans.

A wide range of resistance to curly top was found in the common beans. Among the white beans only 4.7 per cent were found in the highly resistant Grade 1. Among the mottled beans 20 per cent were included in Grade 1, but in the red and pink varieties 35 per cent were found highly resistant. In Grade 5, which includes those beans that were destroyed by curly top, the percentages were as follows: white varieties, 52 per cent; pink and red, 29 per cent; and mottled, 30 per cent.

CONCLUSIONS

1. Curly top of sugar beets attacks varieties of all the commercial species of beans.

TABLE 2.—*Summary of resistance to curly top*

Grades of resistance	1		2		3		4		5	
	Immune or no visible damage		Slight injury		Considerable		Heavy injury		Destruction	
	No. of varieties	Percentage	No. of varieties	Percentage	No. of varieties	Percentage	No. of varieties	Percentage	No. of varieties	Percentage
P. vulgaris White	3	4.7	3	4.7	9	14.0	16	25.0	33	51.6
Pink or Red	5	35.6	2	14.2	2	14.2	1	7.0	4	29.0
Mottled	2	20.0	3	30.0	1	10.0	1	10.0	3	30.0
P. multiflorus White			1	100.0						
P. lunatus sieva White	0	0.0	1	17.0	4	66.0	1	17.0		

FIG. 6. California Pink. Resistant to curly top.

2. Highly resistant varieties were found in the species of common beans and in the small Limas.

3. Resistance to curly top is partially but not exclusively correlated with pink and red colors. White beans, highly resistant to curly top, have been found.

4. A combination of resistance to both curly top and mosaic is possible in a single variety of beans.

LITERATURE CITED

(1) CARSNER, E. Susceptibility of various plants to curly top of sugar beet. Phytopath. **9**: 413–421. 1919.

(2) ————. Attenuation of the virus of sugar beet curly-top. Phytopath. **15**: 745–758. 1925.

(3) ————. Susceptibility of the bean to the virus of sugar beet curly top. Jour. Agr. Res. **33**: 345–348. 1926.

(4) SEVERIN, HENRY H. P. Additional host plants of curly top. Hilgardia **3**: 595–636. 1929.

(5) ————, and C. F. HENDERSON. Some host plants of curly top. Hilgardia **3**: 339–392. 1928.

PHYTOPHTHORA ARECAE, PARASITIC ON ARECA TOPS, AND A STRAIN OF P. PALMIVORA BUTL. (P. FABERI MAUBL.) ON A NEW HOST, ALEURITES FORDI

S. V. VENKATARAYAN

INTRODUCTION

Phytophthora arecae. Towards the end of the year 1929 a certain amount of killing of the tops of the Areca palm, *Areca catechu* L., were reported from the Marthur Experimental Garden in the Mysore malnad, or the portion adjoining the western ghats. The garden is a small one of about 5 acres comprising 2,000 trees. Investigation showed the killing to be due to a species of Phytophthora, and it seemed desirable to see whether this was the same species as that affecting the nuts, namly, *Phytophthora arecae* (Colem.) Pethyb. It was with this purpose in mind that the present investigation was taken up.

HISTORICAL

Phytophthora arecae has been known to affect the Areca palm in two ways: the one resulting in the fall of the nuts, and the other in the death of the tops, or bud rot. Coleman (2) describes two methods of infection of the tops, one by means of the stalks of the bunches of fruits and the other by means of leaf sheaths surrounding the growing point. He found the first method to be more usual than the second but showed by inoculation experiments that the second method was a possibility. The attack on the tree tops, however, was rare, and he accounts for this by the possible washing off of the zoospores by the heavy rains of the monsoon. He does not mention the time of year, if any, when the dying of the tops is at its worst.

Butler (1), working on the bud rot of palms in India, records the fact that, according to popular opinion and his own observations, there is a seasonal prevalence of the disease. He says that the number of cases of infection is greater during the period August to February than during March to July and that humidity seems to be a very important factor for infection. Similarly, Gadd (4), working on the bud rot of Areca in Ceylon, mentions the occurrence of the disease in the month of December. Our experience goes to show that there is a seasonal prevalence of the disease on Areca nut, also. In the Marthur Farm approximately 40 trees died between the months of October, 1929, and February, 1930, while previous to this and even later, no cases were reported. Again, it was only in December, 1930, that the first case of infection for the year was reported from the same place. Another interesting observation by Butler (1) is that

the infection may remain latent for 2 years so that unless scrupulous care is taken in destroying all infective material from the garden and its surroundings, the infection may recur after the lapse of some time.

FIG. 1. The top of an Areca palm showing the infection of the leaf sheaths. The Phytophthora growth may be seen both at the base and the top of the outer sheath as well as in the second sheath in between the margins of the outer sheath.

SYMPTOMS

The outer sheaths of infected tops revealed the presence of mycelium and sporangia of the Phytophthora type, and hyphae were found also in the cells of the infected tops. Both the types of infection described by Coleman (2) were found, those in which the tops showed a watery and slightly discolored appearance at the base of the flower stalks and those in which the bases of the outer leaf sheaths were rotten and could be pulled out quite easily. (Fig. 1.)

ISOLATION

Aseptic pieces of the infected leaf sheaths and the portions at the junction of the discolored and apparently healthy tissue in the tree top, planted in poured plates of malt agar, yielded a fairly pure growth of Phytophthora. The fungus grows well on Quaker-oats agar and French-bean agar but not so well on malt agar. Subsequent transfers were all made to Quaker-oats agar.

THE FUNGUS

Sporangia were rather scanty on the solid media but formed within 24 hours when transferred to a Petri dish containing sterile distilled water. They averaged 38.5 x 29.5 μ (average of 80 measurements). The extremes were 26.4 x 22.2, 55.5 x 42.0, and 62.7 x 39.0 μ. It is interesting to compare these measurements with those given by Coleman (2), which are 30.1 x 20.6, 51.2 x 45.4, and 71.0 x 43.3 μ. No oospores have yet been found in any of the cultures of this fungus from January, 1930, when culture work was commenced. In December, 1930, and January, 1931, chlamydospores were noticed in a subculture transferred in October, 1930. A subculture made in December of the same year showed the formation of chlamydospores in about 20 days. Transfers made prior to October also showed a few chlamydospores when examined after this date, although, during previous examinations, they were never seen. They were not so numerous as they commonly have been in cultures of *Phytophthora Faberi* Maubl., and, as far as we now can judge their production seems to be seasonal. The chlamydospores possess thick walls. They have 3 walls (Fig. 2, D), as seen by Dastur (3) for *P. parasitica* Dastur; the outermost is thin and hyaline, the central thick, and the innermost thicker than the outermost but thinner than the central. The contents are finely granular, with no indication of oil globules. They measured from 24–42 μ, with an average of 34.3 μ (average of 19 measurements).

Oospores in mixed cultures. As oospores were not formed in any of the pure cultures of this fungus it was decided to attempt paired cultures with single-spore strains of the Areca and the sandal Phytophthora isolated by

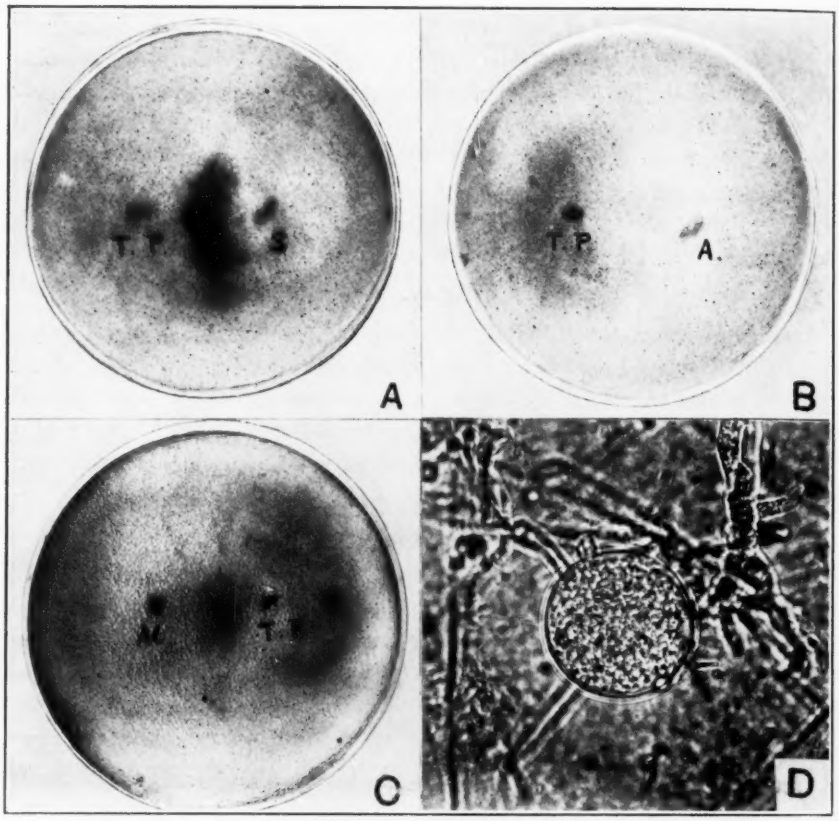

FIG. 2. A. Paired culture of the top-rot Phytophthora (T.P.) and Santalum strain (S). Five-days-old culture on French-bean agar, lighted from underneath. Shows brown line in the middle, which is the area of the greatest development of oospores. B. Paired culture of the top-rot (T.P.) and Areca nut (A) strains. Five-days-old culture on French-bean agar, lighted from underneath. No brown line was formed showing the identity of sex in the 2 strains. C. Paired culture of the top-rot (T.P.) and Aleurites (Al) strains. Seven-days-old culture on French-bean agar, not lighted from underneath. Shows brown line. Compare with figure 4, C. D. Photomicrograph of a chlamydospore of the top-rot Phytophthora. ×600.

Narasimhan (7). In mixed cultures of the fungus from the Areca-tops strain, oospores were formed with the sandal (Fig. 2, A) but none with the Areca strain (Fig. 2, B).[1] Oospore formation was always accompanied by the appearance of the characteristic brown line at the junction of the 2 mycelia, as observed by Narasimhan (7) for his heterothallic strains. The antheridia were all of the amphigynous type. The oogonia made their

[1] I have to thank Mr. M. J. Narasimhan for kindly allowing me to use his isolations of Phytophthora from Areca and sandal in the paired cultures.

appearance sometimes on the second day and often on the third day. They first formed at the meeting line of the 2 mycelia and later appeared on both sides of this line where the hyphae interlaced. Oospores also developed in mixed cultures with the Phytophthora from *Aleurites fordi* Hemsl., described below (Fig. 2, C). Oospores were formed when the pairs were grown in potato-dextrose and carrot agars, but only slowly and without the accompaniment of the brown line at the junction of the mycelia. The diameters of oogonia and oospores and their relation to those given by previous workers are given in table 1.

TABLE 1.—*Measurements of oogonia and oospores of Phytophthora arecae*

	Oogonia	Oospores	Number of measurements
	(diameter in μ)	(diameter in μ)	
Top-rot Phytophthora plus P. on sandal	28.9	25.5	40
P. arecae plus P. on sandal	32.6	28.1	25
P. arecae plus P. on sandal (Narasimhan) (7)		30–31	
P. arecae (Coleman) (2)		23–36	
P. arecae (Rosenbaum) (8)		29.45–31.49	

DISCUSSION

Gadd (4), working on *Phytophthora arecae* causing bud rot of Areca palms in Ceylon, did not obtain oospores either in pure culture or in mixed cultures with any of the 7 strains of *P. Faberi* and with *P. palmivora* from coconut. He found in old cultures spherical bodies resembling chlamydospores produced less abundantly than in *P. Faberi.* In this respect the bud-rot Phytophthora he had under observation seems to agree with that found in Mysore. The diameter of these bodies he gives as 24–48 μ. Chlamydospores have not been noticed in *P. arecae* either by Coleman (2) or Rosenbaum (8). This absence of chlamydospores in *P. arecae,* together with the fact that both Coleman and Rosenbaum found oospores in this fungus, led Gadd to presume that the bud-rot fungus in Ceylon might not be *P. arecae* but *P. Faberi, P. palmivora,* or a new allied species, unless chlamydospores should be found in *P. arecae* under certain circumstances. Narasimhan (7) has suggested that *P. arecae* might be heterothallic and that the male and female strains might possibly tend to get isolated on different hosts by the loss of one or other of the sex strains. If such be the case, it is possible that in Ceylon, where *P. arecae* is a much later introduction than in Mysore, there is only one of the sexes, and, to compensate for the loss of sex, the fungus assumed the ability to produce vegetative resting spores in the form

of chlamydospores. Now that chlamydospores have been noticed in cultures of *P. arecae* producing bud rot in Mysore and that this fungus behaves in respect to heterothallism in the same manner as the strain on the Areca nut, from which *P. arecae* was originally described, Gadd's supposition about the identity of the bud-rot fungus seems to me to be no longer warranted. A more important reason is that Coleman (2) gives as the habitat of *P. arecae* the bases of leaves, fruits, peduncles, and apex of *Areca catechu*, while Gadd (4), himself, says that the fungus from the bud tissues and that from the nuts are indistinguishable in culture.

PHYTOPHTHORA ON ALEURITES FORDI

INTRODUCTION

Towards the end of October, 1930, seedlings of *Aleurites fordi* Hemsl.[2] (the tung-oil tree of commerce) were found to bear leaf spots (Fig. 3). Infected leaves wilted gradually and finally dropped down, leaving the seedling bare of all leaves in extreme cases. In the early stages the leaves show water-soaked areas, either near the margin or the tip, and the spots

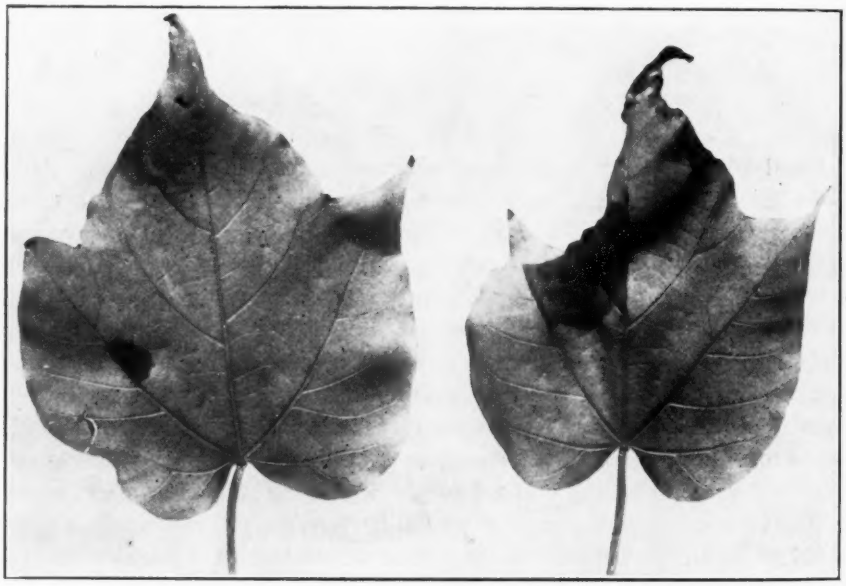

FIG. 3. Two leaves of *Aleurites fordi* Hemsl. showing natural infection due to Phytophthora.

[2] These seedlings were raised by Mr. M. G. Venkata Rao, Assistant Conservator of Forests on sandal-spike duty, from seeds that were obtained from Kew. They were about 4 months old at the time of infection and were growing in pots in the compound of the agricultural laboratories at Bangalore.

later become brown. They are at first small, limited by the prominent veins, and gradually spread and involve almost the whole leaf. Phytophthora mycelium and sporangia were noticed on the under side of affected leaves. This Phytophthora on *A. fordi* is interesting in that this is the first time, as far as can be made out from the literature, that it has been noticed on this host. There are other members of the family Euphorbiaceae, to which Aleurites belongs, that are attacked by Phytophthora, such as *Ricinus communis* L. (3), *Hevea brasiliensis* Muell. (5, 6), *Manihot Glaziovii* Muell., capable of artificial infection only by *P. Meadii* McRae (5) and *Jatropha curcas* L. (7, 9).

ISOLATION

Affected leaves in the early stages of attack were selected and washed well under the tap. Pieces from two of these leaves involving the apparently healthy and the discolored portions were washed in sterile distilled water and transferred to Petri dishes containing malt agar. The resulting growth was pure, and transfers were made to Quaker-oats agar in tubes.

THE FUNGUS

The mycelium grew very well on common media, such as Quaker-oats agar and French-bean agar, there being plenty of aerial mycelium. On potato-dextrose agar and malt agar the fungus did not grow so well as on the above-named media. The growth on carrot agar differed slightly from that on others and from the growth of the top-rot Phytophthora on this same agar (Fig. 4, C). The Aleurites strain showed concentric rings on the carrot agar, with a little spreading growth around the inoculum, whereas the top-rot Phytophthora showed only a spreading growth. In the other media the Aleurites strain showed only a spreading growth. Sporangia developed on Quaker-oats agar but more readily in sterile distilled water in Petri dishes. They measured on the average 32.0 x 23.6 μ (average of 35 measurements), with extremes of 54.6 x 32.7 and 20 x 15 μ. Chlamydospores were formed in large numbers on Quaker-oats agar. They were both terminal and intercalary. They contained a lot of oil globules unlike those of *P. arecae* from the tops (Fig. 4, D). Oil globules have been noticed by Dastur (3) in the chlamydospores of *P. parasitica*. The chlamydospores have 3 walls, the outer very thin and transparent, the central thick and yellowish, and the inner one, which is thinner than the middle one but thicker than the outermost. Dastur (3) says that the outer wall can be seen only when stained and that the innermost wall is not closely attached to the central, thick wall. In the cases observed by me the innermost wall was found closely attached to the central wall, and there was no necessity to stain the chlamydospores to see the outermost hyaline wall, as may be

seen from the photograph, which is of an unstained preparation (Fig. 4, D). They measured on the average 30.9 μ (average of 49 measurements) ranging between 15 μ and 43.8 μ.

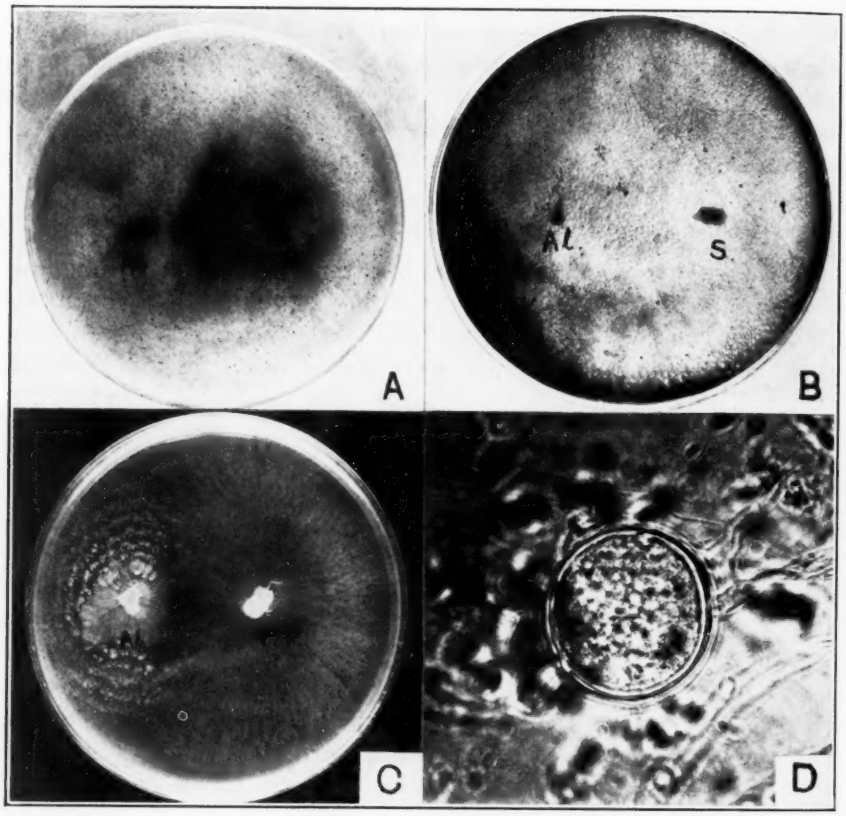

FIG. 4. A. Paired culture of the Aleurites Phytophthora (Al) and Areca nut (A) strain. Five-days-old culture on French-bean agar, lighted from underneath. Shows brown line of oospore region. B. Paired culture of the Aleurites Phytophthora (Al) and Santalum (S) strains. Nine-days-old culture on French-bean agar, not lighted from underneath. No line seen showing the identity of sex in the 2 strains. C. Paired culture of the Aleurites Phytophthora (Al) and top-rot Phytophthora (T.P.). Nine-days-old culture on carrot agar. Oospores developed in 7 days without the accompaniment of the brown line. Note the difference in the mycelial growth of the 2 strains on this agar and compare with figure 2, C. D. Photomicrograph of a chlamydospore of the Aleurites Phytophthora. ×600.

Oospores. Oospores have not been found in cultures nor in the affected leaves thus far examined, but they develop readily in 3 days in mixed cultures on French-bean agar with *Phytophthora arecae* (Fig. 4, A) and

the Phytophthora of the top rot of Areca (Fig. 2, C). In all these cases the characteristic brown line was formed. No oospores were formed in mixed cultures with the Phytophthora on sandal leaves (Fig. 4, B). The antheridia were all of the amphigynous type. The following table shows the measurements of the oogonia and oospores and their relation to the oospores formed in the heterothallic *P. arecae*.

TABLE 2.—*Average diameters of oogonia and oospores in mixed cultures of the Aleurites Phytophthora and P. arecae*

	Oogonia	Oospores	Number of measurements
	(diameter in μ)	(diameter in μ)	
Aleurites Phytophthora plus *P. arecae*	27.8	23.04	25
Aleurites P. plus top-rot strain	25.8	22.2	40
Top-rot P. plus sandal strain (writer)	28.9	25.5	40
P. arecae plus sandal strain (writer)	32.6	28.1	25
P. arecae plus sandal strain (Narasimhan) (7)		30–31	
P. arecae plus Jatropha strain (Narasimhan) (7)		26–27	

It will be seen that the oospores in mixed cultures of the Aleurites Phytophthora and *P. arecae* are smaller than those formed in mixed cultures of the Areca and sandal strains of *P. arecae*, and more nearly approach in size those formed between the Areca and Jatropha strains of *P. arecae* as given by Narasimhan (7).

INFECTION

A preliminary infection experiment was carried out with *Aleurites fordi* seedlings in a moist chamber in the laboratory. One leaf was inoculated on the under surface and another on the upper with 2 loopfuls of zoospore suspension. The check was inoculated in the same manner with sterile distilled water. On the third day the leaf inoculated on the under surface showed dark patches around the inoculated point, while that inoculated on the upper surface and the check remained healthy during the course of the experiment, which lasted 7 days. The fungus was reisolated from the inoculated leaf. It showed the same characteristics as the original isolation. Chlamydospores were first formed in 10 days, increasing in abundance in 17 days to 3 weeks.

RELATIONSHIPS

The abundance of chlamydospore formation in the cultures of the Aleurites Phytophthora leads me to think that it is *Phytophthora palmivora* Butl. (*P. Faberi* Maubl.). Since it forms oospores readily with the Areca strain of *P. arecae*, which has been definitely shown to be the male of a heterothallic species by Narasimhan (7), we may presume that the Aleurites strain is a female, probably of a different species. The oospores look like hybrids, as may be judged by their size (Table 2). The condition resembles that seen when cultures of *P. parasitica*, which have lost their capacity to form oospores individually, are mated with *P. arecae*, when oospores intermediate in size between those of *P. parasitica* and *P. arecae* are formed, as mentioned by Narasimhan (7). In this connection we may compare the mycelial growth of *P. arecae* and the Aleurites strain in French-bean agar and carrot agar (Figs. 2, C, and 4, C). So this fungus on Aleurites probably is not *P. arecae* but a strain of *P. palmivora* Butl. (*P. Faberi* Maubl.).

I am greatly indebted to Dr. Leslie C. Coleman, M.A., Ph.D., Director of Agriculture, Mysore, and Mr. M. J. Narasimhan, Officiating Mycologist, for several helpful criticisms and suggestions during the course of this work.

SUMMARY

Phytophthora arecae was isolated from dying Areca tops. The fungus formed oospores in mixed cultures with the heterothallic strain on *Santalum album* but not with that on the Areca nuts, hence proving its identity with the latter strain.

Chlamydospores have been discovered in the cultures of the fungus of the Areca tops in Mysore, as has already been done for the bud-rot Phytophthora in Ceylon.

A strain of *Phytophthora palmivora* (*P. Faberi*) has been noticed on a hitherto undescribed host, namely, *Aleurites fordi*, the tung-oil tree. In mixed cultures it seems to hybridize with the male strain of *P. arecae*.

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LITERATURE CITED

1. BUTLER, E. J. The bud-rot of palms in India. India Dept. Agr. Mem., Bot. Ser. 3: 221–280. 1910.
2. COLEMAN, L. C. Diseases of the Areca palm. I. Koleroga. Mysore. Dept. Agr. Myc. Ser. Bull. 2. 1910.
3. DASTUR, J. F. On Phytophthora parasitica n. sp., a new disease of the castor-oil plant. India Dept. Agr. Mem., Bot. Ser. 5: 177–231. 1913.

4. GADD, C. H. The relationship between the Phytophthoreae associated with the bud-rot diseases of palms. Ann. Bot. **41**: 253–280. 1927.
5. MCRAE, W. Phytophthora meadii n. sp. on Hevea brasiliensis. India Dept. Agr. Mem., Bot. Ser. **9**: 219–273. 1918.
6. ————. A disease of the Para rubber tree, caused by Phytophthora meadii, McR. Agr. Jour. India **14**: 566–577. 1919.
7. NARASIMHAN, M. J. Studies in the genus Phytophthora in Mysore. I. Heterothallic strains of Phytophthora. Phytopath. **20**: 201–214. 1930.
8. ROSENBAUM, J. Studies of the genus Phytophthora. Jour. Agr. Res. **8**: 233–276. 1917.
9. RUTGERS, A. A. L. Hevea-Kanker III. Mededeelingen van het Laboratorium voor Plantenziekten. No. 28. Dept. v. Landb. Nijverheid en Handel. 1917.

AREOLATE MILDEW OF COTTON

JOHN EHRLICH[1] AND FREDERICK A. WOLF

In 1890 Atkinson (1) briefly described a disease of cotton found by him in Alabama, the causal organism of which he designated *Ramularia areola* Atk. All subsequent reports of this disease by various investigators have dealt primarily with its occurrence and the probable losses caused by it, but nothing has been contributed toward a better understanding of the morphology and development of the pathogen. Accordingly, it was deemed advisable to make such an investigation, and the present paper is the result of these studies.

NAMES

This disease has been given various names, including "areolate mildew" by Atkinson (1), "frosty blight" and "areolate mildew" by Duggar (6, page 296), "mildew" by Cook (5), "false mildew" by Vincens (21), and "gray mildew" by Laycock (14). Of these, areolate mildew appears to be preferable and, accordingly, is used in this report.

SUSCEPTS

Since no effort has been made by any one to determine the suscept range, there are no data on this point. The pathogen is not known to occur, however, except on species of Gossypium.

The organism was first reported on the leaves of cultivated upland cotton, *Gossypium hirsutum* L. It is not confined to this species, however, since Farquharson (7) has observed that American cotton in Southern Nigeria is very commonly attacked, while it does not seriously affect such native cottons as *G. peruvianum* Cav. and *G. vitifolium* Lam. Vincens (21) reported that the fungus occurred on imported varieties of cotton in the Botanical Gardens of Saigon in Indo-China but left the indigenous varieties practically unharmed. Sea-island cotton, *G. barbadense* L. also is known to be a suscept.

IMPORTANCE AND RANGE

Areolate mildew is a disease of the enphytotic type, which may be found every season practically wherever cotton is cultivated. In spite of this, the actual losses from it are slight or inappreciable, except on rare occasions and in limited localities. Usually it involves only that part of the crop that is situated in low-lying portions of the field.

Since the discovery of areolate mildew near Auburn, Alabama, it has

[1] This investigation was conducted while the senior writer held a graduate fellowship at Duke University, Durham, N. C.

been collected, according to the records of the Division of Mycology and Disease Survey of the Bureau of Plant Industry, United States Department of Agriculture, in the following Cotton Belt States: Louisiana, Mississippi, Florida, Georgia, South Carolina, North Carolina, Tennessee, and Arkansas. There are, in addition, various reports from the West Indies.

The first collection in South America was made by Spegazzini[2] in Paraguay and Brazil in 1892. He described the pathogen as *Cercosporella Gossypii* Speg. A report from São Paulo, Brazil (15), states that cotton leaves there are attacked by *Ramularia aérea,* presumably, an incorrect spelling of *R. areola* Atk.

Birmingham and Hamilton (4), in 1923, noted the occurrence of areolate mildew in Australia. The disease has been reported for a number of years from various parts of Africa. Farquharson (7), in 1914, noted that it was very commonly present on American cotton in Southern Nigeria, as previously stated. Two years later, Small (16) recorded its occurrence in Uganda, chiefly on young plants. His later reports (17, 18) state that mature leaves of cotton are severely attacked, especially during prolonged wet periods. Others who have recorded its occurrence here include Snowden (19), Laycock (14) and Hansford (8), the latter of whom stated that it was very commonly present but that it caused little damage.

Wallace (22) observed areolate mildew in Tanganyika and Swainson-Hall (20) in West Africa, and various collections have been made in the Belgian Congo.

In Asia the records of Vincens (21) show its occurrence in Cambodia and in the Botanical Garden of Saigon.

SYMPTOMS

Areolate mildew usually appears on the leaves toward the close of the growing season and may be distinguished readily from all other cotton diseases. Its lesions are irregular, angular areas bordered by the veinlets and are light green to yellowish green when viewed from the upper surface. They have the appearance of being covered by mildew when seen from the lower surface, due to the profusion of conidiophores and conidia (Fig. 1). Occasionally the white coating appears also on the upper side. Lesions of similar appearance may form on the bracts surrounding the bolls.

The defoliation, which may follow from abundant lesions on the leaves, may result in the premature opening of the bolls and the consequent deterioration of the staple, as has been suggested by several observers.

THE PATHOGEN

The present studies show that the fungus that causes areolate mildew possesses three stages in its cycle of development, a conidial stage, described

[2] Saccardo, P. A. Sylloge Fungorum 10: 565. 1892.

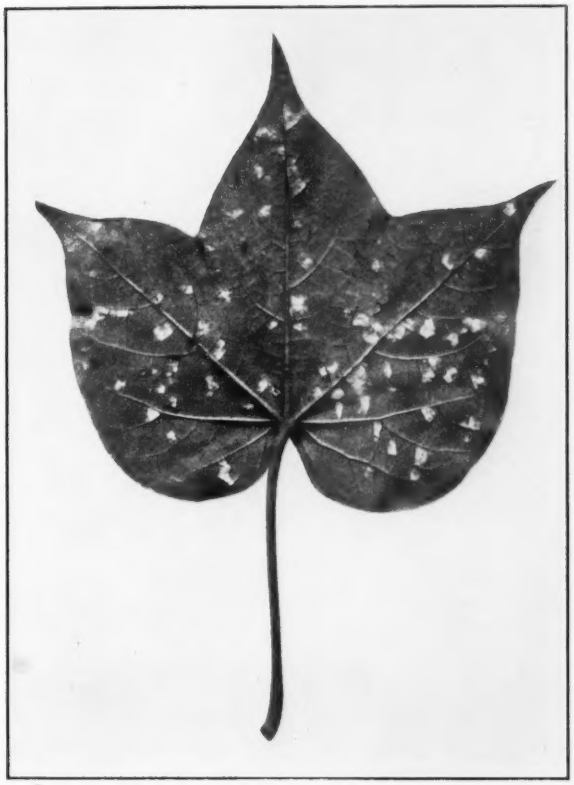

FIG. 1. Areolate mildew of cotton. (Courtesy of W. W. Gilbert).

by Atkinson (1), in 1890, as *Ramularia areola* and as *Cercosporella Gossypii* by Spegazzini in 1892, which appears on the living tissue; a spermogonial stage, which occurs in autumn on the fallen leaves; and an ascigerous stage, which develops in the spring on decaying leaves.

Conidial stage. Microscopic examination shows that clusters of hyaline conidiophores, 25 to 75 × 4.5 to 7.0 μ, occur on the lower surface of lesions, rarely on both surfaces. They bear 1- to 3-septate, hyaline conidia, 14 to 30 × 4 to 5 μ (Fig. 2, E, F), either terminally or laterally, which may remain in chains or occur singly.

Paraffin sections show that the conidiophores emerge through the stomata and arise from a loosely compacted, substomatal stroma. The fascicle is so constricted at the stomatal orifice as to look like an hourglass (Fig. 2, A). Hyphae ramify from the stroma, coursing both between and within the spongy and palisade parenchyma, and may so closely intertwine with those radiating from neighboring centers as to form an almost continuous stroma within the limits of a single lesion.

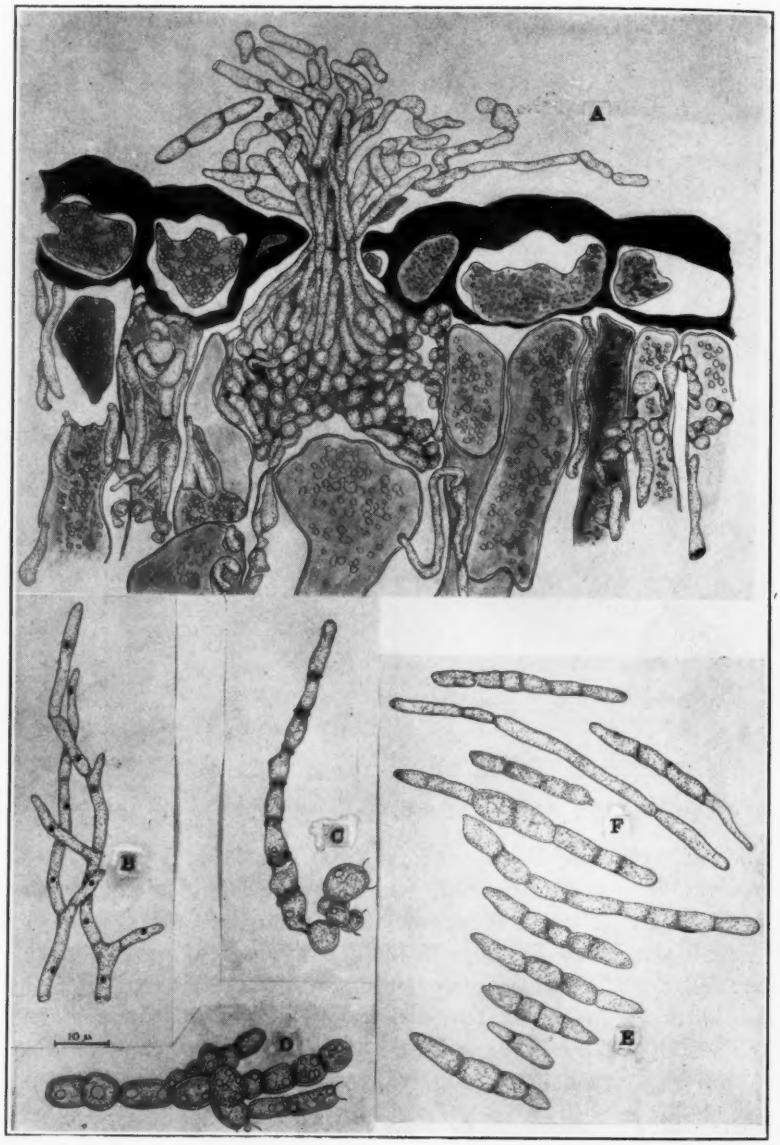

FIG. 2. A. Upper surface, in vertical section, of cotton leaf affected with areolate mildew. The conidiophore fascicle arises from a substomatal stroma and extends to the surface through the stomatal orifice; mycelium both between and within the palisade parenchyma. B. Aerial mycelium from culture. C. Hyphae with plate-like septa at the surface of the agar. D. Hyphae from beneath the surface of the agar. E. Conidia. F. Germinating conidia.

A destruction of invaded cells accompanies the advance of the mycelium. In newly attacked cells the protoplasm becomes more granular and is later plasmolyzed. The middle lamella is manifestly dissolved, as shown by the separation of suscept cells. The cells finally collapse completely and disintegrate, leaving only fragments of the secondary membranes.

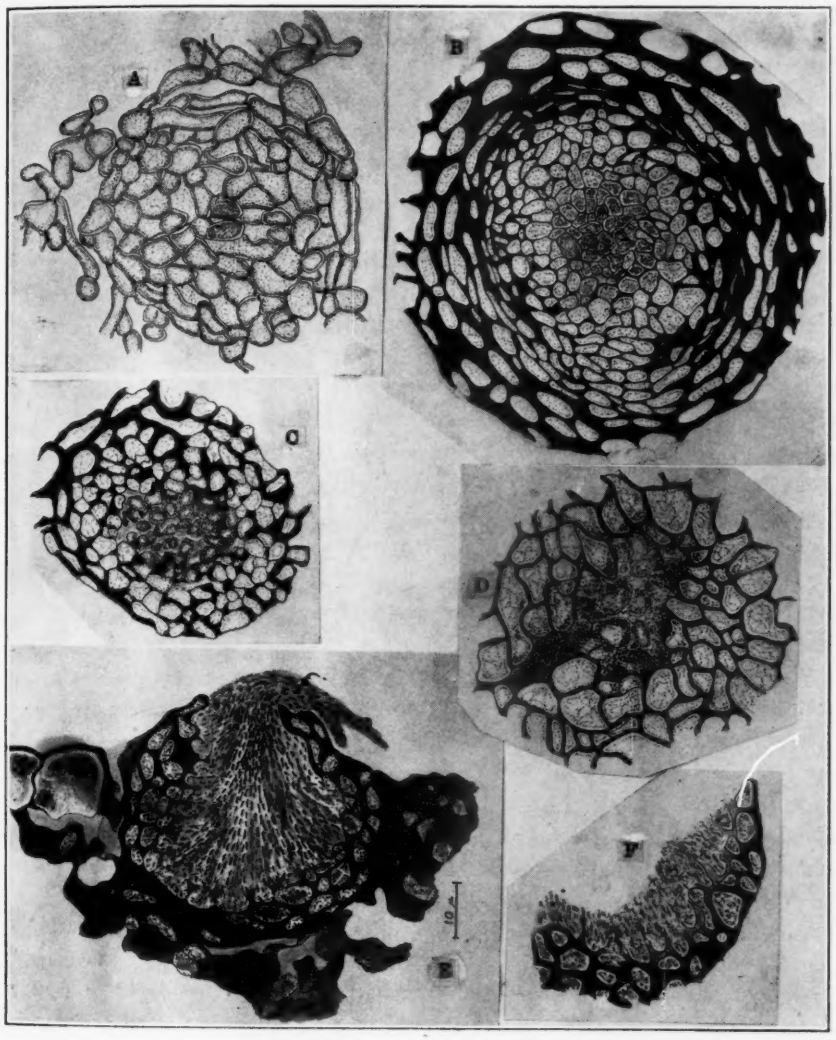

FIG. 3. A. Median section of young sclerotium in culture, an early stage in formation of spermogonium. B. The walls of the cortical cells of the developing spermogonium have become thick and those of the medullary portion densely filled with protoplasm. C and D. Early stages in formation and liberation of spermatia. E. Mature spermogonium in vertical section with spermatia embedded in a mucilaginous matrix. F. Section of the spermatiferous area of the inner wall of a spermogonium.

Collections of leaves bearing an abundance of conidia were made in October, 1930, and placed in wire baskets out of doors. In April, 1931, a whitish coating of conidia was present on same of these. When these conidia were placed in water, they germinated, thus showing that viable conidia are at hand in the following growing season.

Spermogonial stage. By the time the leaves have withered and fallen and becomes brown, the lesions appear as sharply defined, darker areas. An examination of the lower surface with a hand lens reveals numerous slightly raised black spots, the spermogonia. They are found to be mature throughout November and December. They measure 28 to 75 μ in diameter, and, when crushed on a slide under a cover-glass, numerous spermatia, 2.4 to 4.0×0.4 to 2.0 μ, ooze out. Attempts to induce germination of spermatia met with consistent failure.

Sections of affected tissues during this period show various stages in the development of spermogonia. At an early stage they are sclerotium-like bodies composed of compact thick-wall cells (Fig. 3, A). The walls of the innermost cells, which are the first to become spermatiferous, next become thinner and their cell content more densely granular and deep-staining (Fig. 3, B). Each protoplast eventually becomes separated into a number of rod-shape or bone-shape spermatia, after which the cell wall disintegrates and the spermatia are liberated in a matrix consisting of cytoplasm and disintegrating cell walls (Fig. 3, C, D, F). This form of spermatia and their liberation proceed centrifugally until only the outer layers of thick-wall cells remain, forming the spermogonial wall. With the apical opening of the spermogonium the spermatia are exuded in a viscous mass through the ostiolum (Fig. 3, E).

This endogenous formation of spermatia (variously designated pycnospores, microconidia, and stylospores) has been previously described, apparently, only by Laubert (13) for an unnamed organism occurring on dying apple branches and by Klebahn (12) for *Mycosphaerella Hieracii* (Sacc. et Br.) Jaap. Laubert states: "die Sporen dadurch entstehen, dass sich die Zellinhalte des anfangs vorhandenen, farblosen Paraplechtenchyms zu dünngleichzeitig die Membranen der Mutterzellen verquellen und vergallerten." Klebahn describes the origin of "Mikroconidien," "als ob sie durch Zerfall der Hyphen eines feinfädigen Geflechts, das das Innere der Sklerotien, vermischt mit weiteren Zellen, auffüllte, entstanden waren."

Ascigerous stage. The perithecia are sparingly present in April on the lower leaf surface in the areas previously occupied by the conidia and spermogonia. They have been noted in each of the three seasons in which this organism has been studied. They appear to arise from stromatic or sclerotial structures embedded within the tissues of the leaf. These

sclerotia occur in abundance throughout the fall, winter, and spring. They are first noted among the spermogonia, and no means has been determined of knowing which of these are destined to become spermogonia or perithecia or which are to remain as sclerotia.

Mature perithecia are dark brown, with a slight papilla, and measure 70 to 80 μ in diameter. They project well from the leaf surface (Fig. 4, A).

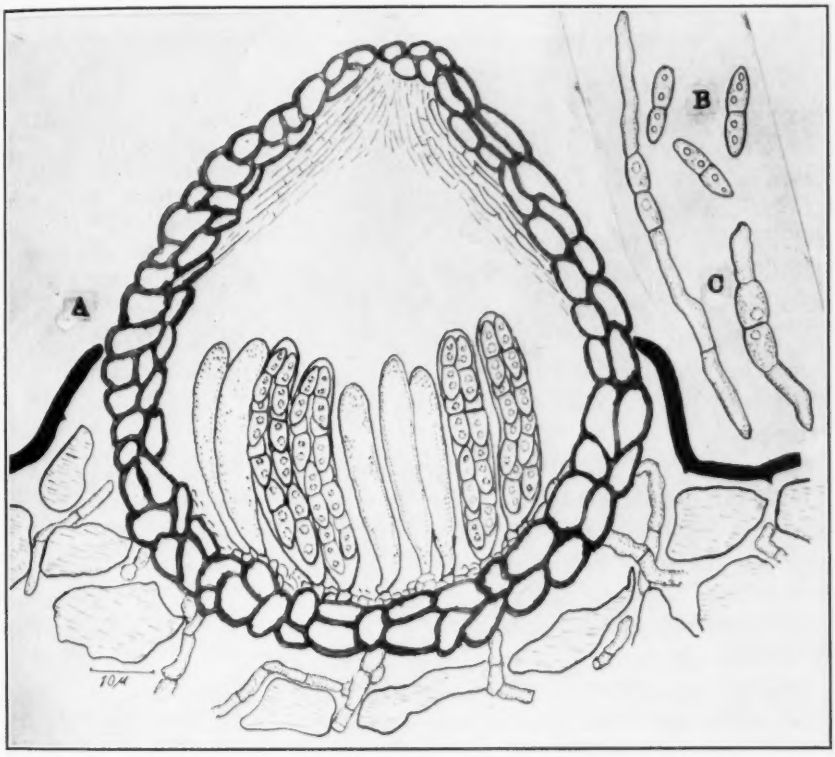

FIG. 4. A. Median section of a perithecium of *Mycosphaerella areola*. B. Ascospores. C. Germinating ascospores.

The asci are 35 to 40 × 6 to 8 μ, and the biseriate ascospores are elongated and slightly constricted at the septum, which divides the spore unequally (Fig. 4, B). They measure 12.4 to 15.6 × 3.2 to 3.8 μ.

Growth in culture. Attempts to isolate the pathogen in pure culture by the use of conidia led to the conclusion that it is a difficult procedure. In the greater proportion of trials, the conidia failed to germinate when placed in tap water or distilled water, even when bits of cotton leaves or traces of sugar were added. In some cases a small proportion had begun to grow after 8 to 10 hours. When such germinating conidia (Fig. 2, F) were

spread over the surface of cotton-leaf-decoction agar, slow-growing, grayish white, hemispherical colonies, 1 to 3 mm. in diameter, appeared after several weeks. The aerial mycelium was slightly fluffy and consisted of slender, thin-wall hyphae of uninucleate cells (Fig. 2, B). At the level where the aerial hyphae enter the substratum, the walls thicken, and the septa appear as plates, which extend beyond the lateral walls (Fig. 2, C). Within the substratum the mycelium is very compact and is made up of rounded thick-wall cells filled with oil globules (Fig. 2, D). In a series of cultures planted on October 23, 1928, both conidia and spermogonia bearing spermatia had formed by December 15. These conidia were like those present on leaf lesions. Paraffin sections of such colonies on cotton-leaf-decoction agar show that spermogonial development in agar is of the same type as occurs on leaves.

Isolations from ascospores were made by inverting agar plates over decaying leaf fragments, placed on moist blotting paper in the tops of Petri dishes. The ascospores, which were forcibly ejected and were lodged on the surface of the agar, where they had begun to germinate (Fig. 4, C) after 15 hours, were transferred to potato-agar slants. Here they developed into colonies of the same type of growth as those isolated from conidia. They have, however, not been observed to form either conidia or spermogonia.

Pathogenicity. All attempts by means of artificial inoculations to establish the pathogenicity of the areolate-mildew organism have been unsuccessful. Suspensions of conidia, both from leaves and from culture, have been placed by means of a camel's-hair brush on both the lower and upper surface of young and mature, healthy cotton leaves. The inoculated plants were then covered with bell jars or wet cotton was placed on the leaves, after which they were wrapped in wax paper.

Blocks of agar in which ascospores had been discharged were also placed on leaves and protected against too rapid desiccation in the same manner. Failure to secure infection is doubtless due primarily to lack of suitable moisture conditions, as indicated by all observations to the effect that wet seasons are necessary for the occurrence of the disease in the field.

DISCUSSION

Of the approximately 400 species of Ramularia that have been described, the perithecial stage is known for less than a dozen. Killian (10) states that Ramularia is characterized, in general, by the complete suppression of the ascigerous stage and that those species in which it persists produce perithecia rarely or under special conditions, only. Those whose perithecial stage is known belong with one exception to the genus Mycosphaerella. This exception is *Fabraea Ranunculacearum* Fr., which appears in autumn

on *Ranunculus repens* E. & E. Killian (11) showed that its conidial stage is *Ramularia repentis* Oud., in confirmation of the earlier observations of Goussewa, and he noted, in addition, the presence of a pycnidial (spermogonial) stage.

In his earlier studies dealing specifically with *Ramularia Geranii* (West) Fckl., *R. Adoxae* Rabenh., *R. Saxifragae* Syd., *R. variabilis* Fckl., *R. Lapsanae* Desm., and *R. Parietariae* Passer., Killian (9) concluded that most Ramularias produce a second type of reproductive organ, the sclerotium. He regards sclerotia in various stages of development as a transition leading toward perithecia. Some species, as *R. variabilis*, normally produce conidia, spermogonia, sclerotia, and perithecia, and might be properly regarded as complete-cycle forms; others, as *R. Saxifragae*, produce only conidia and sclerotia; still others, as *R. Parietariae* and *R. Geranii*, form conidia, only. The latter of these is known to produce conidia or conidiophores on decaying leaves in the spring. It may be recalled at this point that viable conidia of the cotton Ramularia are present in the spring on conidiophore fascicles. It, therefore, would appear from the studies made on Ramularia that relatively few species bear perithecia and that certain of these, as the fungus here under consideration, produce them only sparingly.

Two other well-known species, *Mycosphaerella Fragariae* (Tul.) Lindau (*Ramularia Tulasnei* Sacc.) and *M. Hieracii* (Sacc. et Br.) Jaap. (*R. Hieracii* (Baumler) Jaap.) have been noted to produce ascospores within the perithecia and conidia at their surface.

While adequate proof of the connection of the conidial stage of the cotton fungus with the perithecial stage is lacking, the evidence in hand warrants one in regarding them, with a good degree of certainty, as stages of the same organism. This evidence may be summarized as follows: (1) Isolations from conidia and from ascospores produce colonies of similar appearance; (2) when infected leaves are allowed to overwinter out of doors an ascogenous stage appears in the spring in the areas previously occupied by the conidial stage and then the spermogonial stage, this having been noted during each of the three seasons during which this study has been in progress: (3) with the exception of *Ramularia repentis*, all species of Ramularia whose ascigerous stage is known have been found to belong to Mycosphaerella.

The perfect stage of the areolate-mildew fungus has apparently not been described previously. Moreover, it is highly improbable that the organism under consideration occurs on species outside the genus Gossypium, since Ramularias are all known to be restricted in their suscept range. The only

Mycosphaerella known to occur on cotton is *M. Gossypina* Atk. (3), type specimens of which were examined.[3]

These specimens, collected in autumn, show both Cercospora and mature Mycosphaerella in abundance in the same lesion. Further, the ascospores of *M. Gossypina* are somewhat larger than and of a different shape from those of the areolate-mildew fungus. For these reasons the name *Mycosphaerella areola* is proposed for the areolate-mildew organism with the following brief description:

Mycosphaerella areola, n. sp.

Syn. *Ramularia areola* Atk.

Cercosporella Gossypii Speg.

Status asciger:

Perithecis hypophyllis, paucis, in maculis aggregatis, parenchymate foliorum immersis, epidermide primo tectis dein suberumpentibus 70–80 μ diam.; asciis fasciculatis, oblongis, 35–40 × 6–8 μ, 8 sporis; sporidiis distichis, oblongis, utrinque obtusiuscle rotundatis, inaequaliter didymis, loculis guttulatus, non vel vix subconstrictis, hyalinis, 12.4–15.6 × 3.2–3.8 μ. Hab. in foliis putridis Gossypii sp.

Status spermatiferus:

Spermogonia dense gregaria in foliis siccis, maculas definite occupans; 28–75 μ diam.; spermatiis hyalinis, 2.4–4.0 × 0.4–2.0 μ.

Status conidicus:

Plagulis superiore primo pallidis inferiore effuso-candidis, angulosis, irregularibus, venulis foliis limitatis, 3–4 mm. diam., saepe confluentibus; hyphis hypophyllis, raro amphiginiis in caespitulos laxe aggregatis, erectis, saepe basi ramosis v. rareus supra denticulatis, pluries septatis, hyalinis, 25–75 × 4–7 μ; conidiis cylindraceo-ellipticis, utrinque saepe abrupte acuminatis, 1–3-septatis, primitus concatentatis, hyalinis, 14–30 × 4–5 μ. Hab. in vivis foliis Gossypii sps.

SUMMARY

Areolate mildew is a common cotton disease of minor importance that occurs on various species of Gossypium, including *G. hirsutum*, *G. barbadense*, *G. peruvianum*, and *G. vitifolium*. It has been observed throughout the cotton-growing areas of the southeastern United States, the West Indies, Africa, Australia, and Indo-China.

Areolate mildew is characterized by the presence of angular, whitish patches, usually on the lower leaf surface, which, as a rule, are first evident toward the close of the growing season.

[3] Examination of type specimens was made possible through the courtesy of Dr. H. M. Fitzpatrick.

The pathogen, known in its conidial stage as *Ramularia areola*, occurs on living leaves in late summer; produces the spermogonial stage on fallen leaves during late autumn; and the ascogenous stage appears in the spring, on decaying leaves. Evidence of the connection of the conidial and the perfect stages is presented.

The sclerotia, from which both spermogonia and perithecia arise, are similar in appearance. The spermatia arise endogenously. The perfect stage is herein described as **Mycosphaerella areola**, n. sp.

LITERATURE CITED

1. ATKINSON, G. F. A new Ramularia on cotton. Bot. Gaz. **15**: 166–168. 1890.
2. ————. Areolate mildew of cotton. *In* (Some diseases of cotton). Ala. Agr. Exp. Sta. Bul. 41: 55–58. 1892.
3. ————. Sphaerella gossypina, n. sp., the perfect stage of Cercospora gossypina, Cooke. Bul. Torrey Bot. Club **18**: 300–301. 1891.
4. BIRMINGHAM, W. A., and I. G. HAMILTON. Diseases of the cotton plant. Agr. Gaz. New South Wales **34**: 805–810, 877–886. 1923.
5. COOK, M. T. Diseases of tropical plants. 317 pp. Macmillan Co., Ltd., London. 1913.
6. DUGGAR, B. M. Fungous diseases of plants. 508 pp. Ginn and Co., New York. 1909.
7. FARQUHARSON, C. O. Plant diseases in Southern Nigeria. Ann. Rpt. Agr. Dept. So. Prov. Nigeria **1913**: 41–55. 1914.
8. HANSFORD, C. G. Cotton diseases in Uganda, 1926–1928. Empire Cotton Grow. Rev. **6**: 10–26 160–167, 240–245. 1929.
9. KILLIAN, CHARLES. Études biologiques du genre Ramularia. I. Partie. Ramularia Geranii (West) Fuck; Ramularia Adoxae Rabenh. Rev. Path. Vég. et Ent. Agr. **10**: 277–302. 1923.
10. ————. Études biologiques du genre Ramularia. Ann. Epiph. **12**: 147–164. 1926.
11. ————. Développement et biologie du Ramularia repentis Oud. Bul. Soc. Mycol. France **45**: 145–152. 1929.
12. KLEBAHN, H. Haupt- und Nebenfruchtformen der Ascomyzeten. 395 pp. Gebrüder Borntraeger, Leipzig. 1918.
13. LAUBERT, R. Ein interessanter neuer Pilz an absterbenden Apfelbäumen. Gartenflora **60**: 76–78. 1911.
14. LAYCOCK, T. Mycology. Ann. Rpt. Agr. Dept. So. Prov. Nigeria **1920–1921**: 23–25. 1922.
15. São Paulo. Serviço de Inspecção e Defesa Agricola. Molestias dos algodeiros, marmeleiros e macieras. (Diseases of cotton, quince, and apple). Bol. Agr. São Paulo **20**: 90–91. 1919.
16. SMALL, W. Annual report of the government botanist for 1914–1915. Ann. Rpt. Dept. Agr. Uganda Protect. **1914–1915**: 57–70. 1916.
17. ————. Annual report of the government botanist for 1919–1920. Ann. Rpt. Dept. Agr. Uganda Protect. **1919–1920**: 42–44. 1921.
18. ————. Annual report of the government mycologist. Ann. Rpt. Dept. Agr. Uganda Protect. **1922**: 27–29. 1924; **1923**: 14–15. 1924; **1924**: 18–20. 1925.

19. SNOWDEN, J. D. Report of the government botanist for the period 1st April to 31st December, 1920. Ann. Rpt. Dept. Agr. Uganda Protect. **1920:** 43–46. 1921.

20. SWAINSON-HALL, R. Diseases of the cotton plant in West Africa. Textile Mercury **69**: 206–207. 1923.

21. VINCENS, F. Rapport sommaire sur les travaux effectués au Laboratoire de Phytopathologie de l'Institut Scientifique de l'Indochine du Janvier 1919, au ler Juliet 1921. Saigon: Imprimérie Commerciale, 19 pp. 1921. (From Bul. Agr. Inst. Sci. Saigon 3: 307–323. 1921).

22. WALLACE, G. B. Diseases of plants. Rpt. Dept. Agr. Tanganyika Territory for the year ending 31st March, **1928:** 40–42. 1929.

SCLEROSPORA GRAMINICOLA ON BAJRA (PENNISETUM TYPHOIDEUM)

H. Chaudhuri

Sclerospora graminicola (Sacc.) Schroet. produces "green-ear disease" of bajra in India. As the name signifies, it is characterized by the reversion of whole or part of the ear into a green, leafy condition. Figure 1 shows a photograph of typical green ears of bajra. This disease is very common in the Punjab and often causes serious damage, as it results in complete loss of grain in the affected plants. It was first described in India by Butler (1). He states that it is usually not of sufficient intensity to attract much notice, though at times, particularly in low-lying, ill-drained lands, it causes considerable damage. Mitter and Tandon (12) have confirmed Butler's observations. The writer, in a paper on green ear of bajra (3), showed that the disease was of considerable importance and that the incidence of the epidemic could not be attributed to either bad drainage or special atmospheric conditions. It occurs frequently in all parts of the Punjab. In recent years it has been observed in many more fields in and near Lahore than in 1926–27.

The first symptom of the disease is shown by young plants long before flowering. They show whitening of leaves in long streaks and, when old, the tissues turn brown, tear along the streaks, and, like the disease in Jowar (*Andropogon Sorghum* Bret.), numerous oospores immersed in the leaf are formed. Badly affected plants do not produce any ear but tufts of leaves instead. Sometimes part of the ear is transformed into twisted narrow leaves (Fig. 1, A), or the whole inflorescence may be shortened and bear only long twisted leaves. The conidial stage of the fungus has not thus far been encountered.

In the Punjab one crop of bajra is raised ordinarily, which is sown in June–July and harvested in October. But sometimes, for fodder purposes, an early crop also may be raised. In September, 1929, the writer found near Lahore a plot of bajra in which all the plants were very badly affected. In that plot not one mature ear was formed. As the plants had not formed any grains, the crop was not harvested at all but was left to stand in the field. On visiting the plot again in early November, the writer found many of the plants dead and, though all of them appeared to be dry and dead, some had developed fresh, leafy, green ears, which came out of the axils of the old ones, now completely dry, and stood out prominently. The development of the green ears twice over in the same plants was no doubt very unusual.

FIG. 1. Two typical green ears of bajra. A. Only half the ear is transformed. B. A completely transformed ear. $\times \frac{1}{3}$.

Diseased plants were brought into the laboratory for study. Sections of every part were cut and examined microscopically. The twisted leaves in the ears did not show any oospores or mycelium, but the ordinary leaves below the inflorescence, which showed white streaks and later shredding, were full of oospores. Figure 2 shows a photomicrograph of a bit of such a leaf. It is full of oospores. The oospores on an average measure 45 μ in diameter. Inoculation experiments with oospores were performed. Twenty young plants of bajra growing in pots were obtained and 15 of these were inoculated with oospores from bajra of the previous season. Five plants were kept as controls. For inoculation a bit of the shredded

leaf was taken and dipped in mercuric chloride solution (1 in 500) for 5 minutes. It was then thoroughly washed in several changes of distilled water, after which the bit of the leaf was tested in a tube containing sterilized distilled water. Loopfuls of spore suspensions were placed on both surfaces of the experimental plants and in a few cases the leaves were also pricked before the spore suspensions were placed. For 24 hours the inoculated plants, as well as the controls, were kept in a chamber of saturated atmosphere. Then all the plants were removed to the glass house in the laboratory. Some of the inoculated plants showed whitening and later shredding in about 3 to 4 weeks' time. No green ears, however, were

FIG. 2. Photomicrograph of a bit of shredded leaf of bajra, showing numerous oospores. ×35.

formed and the control plants continued perfectly healthy. The shredded leaves of the inoculated plants when examined microscopically showed plenty of oospores.

GERMINATION OF OOSPORES

As Butler (1, 2) mentioned germination of oospores from bajra was unknown and its life history in India was not completely known, the writer tried various methods for germinating the oospores. While at the Sorbonne in 1927–28, he tried to germinate oospores collected during the previous season in India. The oospores after surface disinfection were placed on slides containing ample quantities of various liquids, *viz*, water, soil decoction, and various synthetic media. These were placed inside sterilized moist Petri dishes and incubated at different temperatures. Only in isolated cases rupturing of oospore walls and formation of abortive germ tubes took place. But no regular germ tubes developed.

During the last three years Hiura (5, 6, 7) and later, Evans and Harrar (4), following Hiura's method, have succeeded in germinating Sclerospora oospores. The writer has also succeeded in germinating the oospores from

bajra in the same way. This was to place a small piece of moist filter paper bearing minute quantities of oospore powder on the surface of a large bit of moistened cotton in the bottom of a Petri dish (care being taken that the contact between the paper and the cotton be only partial) and a second similar layer in the lid of the dish; the space between the two being about half the height of the dish. Figure 3 shows germination of the oospores

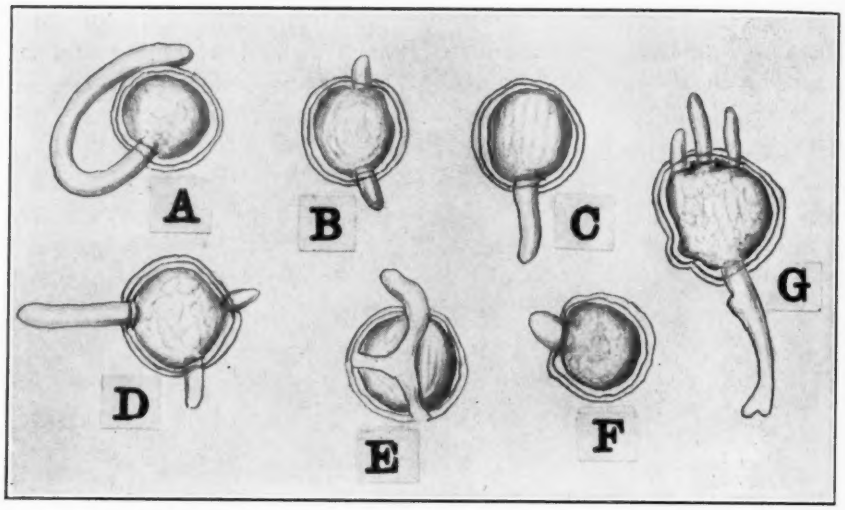

FIG. 3. Germination of oospores. × about 400. A–D. Oospores from 1930 plants. E. From 1929 plant. F. From 1928 plant. G. From 1927 plant.

from bajra. Though Evans and Harrar have sketched germinating oospores with very long germ tubes similar to those described by Hiura, the germ tubes from oospores of bajra Sclerospora were never long. It will be seen that in many cases more than one germ tube developed and figure 3, G, shows an oospore with 4 germ tubes. In germination a portion of the outer coat may be ruptured and the inner coat protrude out as a germ tube or the outer coat may split into several parts before the germ tubes are formed.

VIABILITY OF THE OOSPORES

As the writer had collected and preserved infected bajra leaves every year during the last 5 years, he had the opportunity of studying the period of viability of these bajra oospores. It is interesting to note that even 5-year old oospores did not lose their power of germination. Previously Melhus and others (10, 11) had found Sclerospora oospores viable for 30 months under laboratory conditions. Considering the very thick coat of the oospores, the long period of viability is not surprising.

DISCUSSION AND CONCLUSIONS

Formation of conidial stage in Sclerospora has been reported by many workers. Though the writer has failed to encounter sporangial stages, they have been reported by Butler in India. He found them evanescent and sometimes absent altogether. Kulkarni (9) found the sporangial stages in great abundance on bajra and jowar in Bombay Presidency. The infection was noticeable from the beginning, even when the seedling had put forth only 2 or 3 leaves and conidia were produced only at night. Melhus, Van Haltern, and Bliss (11) observed conidial formation both during night and day whenever certain conditions were fulfilled, *viz.*, (a) complete saturated atmosphere; (b) turgid host tissues; (c) slight moisture film on the surface of the leaves; (d) temperature between 8° and 27° C. Perhaps the failure of the conidial stage of the fungus in the Punjab may be attributed to the absence of a complete saturated atmosphere and also to the absence of a slight moisture film on the surface of the leaves due to very low humidity of the atmosphere in this part of the Punjab.

Regarding infection, Hiura (8) has shown that the disease is of a systemic character, and by inoculation experiments he proved that seedlings may be infected through the roots, coleoptile, and rhizomes. Melhus, Van Haltern, and Bliss (11) found that when oospores overwintered naturally in the field they produced twice as much infection as oospores kept in the laboratory. They showed soil infection took place in almost all cases when plants were grown in untreated soil, but repeated trials on sterile soil gave no evidence to support the possibility of the fungus overwintering in the mycelial stage within the seeds of the host plant. Kulkarni (9) tried inoculation with zoospores in a large number of plants without success. Even when a healthy plant touched a diseased plant in the field, it never got infected. Butler also failed to inoculate healthy leaves with zoospores. These show definitely that in bajra sporangial stages, even though present, are incapable of bringing about any infection, and, as there is no evidence in support of the view that the fungus passed the unfavorable period within the seeds of the host plants, the most plausible explanation is that the fungus overwinters in the soil. As the Sclerospora oospores from bajra have now been germinated and their long viability proved, it would not be wrong to assume that in India the green-ear disease of bajra is perpetuated through the oospores, which are admirably suited to the purpose owing to their very resistant wall.

SUMMARY

Green-ear disease of bajra, caused by *Sclerospora graminicola*, has been studied. It causes considerable loss due to the reversion of the ear into a green, leafy condition. Unusual formation of green ear twice over in the

same plant has been noticed. No conidial stages have been found; only oospores are formed. Successful inoculation experiments, using oospores, have been performed. Though no green ears were formed on the inoculated plants, the leaves showed usual shredding with plenty of oospores. Following Hiura's method, bajra oospores have been germinated. These are found to remain viable even for 5 years. As zoospores are incapable of infecting bajra, this germination of oospores and their long viability and successful inoculation experiments with oospores show beyond doubt that the disease is propagated through the oospores in the soil.

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LITERATURE CITED

1. BUTLER, E. J. Some diseases of cereals caused by Sclerospora graminicola. Mem. Dept. Agr. India, Bot. Ser. 2, No. 1. 1907.
2. ————. Fungi and disease in plants. 547 pp. Thacker, Spink & Co., Calcutta. 1918.
3. CHAUDHURI, H. Green-ear disease of bajra (Pennisetum typhoideum Rich.). Proc. Indian Sci. Congress **15**: 223. 1928.
4. EVANS, M. M., and GEORGE HARRAR. Germination of oospores of Sclerospora graminicola (Sacc.) Schroet. Phytopath. **20**: 993–997. 1930.
5. HIURA, M. Studies on some downy mildews of agricultural plants. I. On Sclerospora graminicola (Sacc.) Schroet., the causal fungus of downy mildew of Italian millet. (The Third Preliminary Note). Agr. & Hort. (Japan) **4**: 525–534 (11–20). 1929.
6. ————. Studies on some downy mildews of agricultural plants. I. On Sclerospora graminicola (Sacc.), the causal fungus of the downy mildew of Italian millet. (The Fourth Note). Reprinted from Boyachu-Gai Zasshi (Jour. Plant Protec. **16**, No. 9, 5 pp. 1929. (Abst. in Rev. Appl. Mycol. **9**: 105. 1930).
7. ————. A simple method of germination of oospores of Sclerospora graminicola. Science, n. s., **72**: 95. 1930.
8. ————. Further note on the downy mildew of Setaria italica. Reprinted from Boyachu-Gai Zasshi (Jour. Plant Protec.) **17**, No. 8. 7 pp. 1930. (Abst. in Rev. Appl. Mycol. **10**: 23. 1931).
9. KULKARNI, G. S. Observations on the downy mildew of bajri and jowar. Mem. Dept. Agr. India, Bot. Ser. **5**: 268–273. 1913.
10. MELHUS, I. E., and F. H. VAN HALTERN. Sclerospora on corn in America. Phytopath. **15**: 720–721. 1925.
11. ————, ————, and D. E. BLISS. A study of Sclerospora graminicola on Setaria viridis (L.) Beauv. and Zea mays L. Iowa Agr. Exp. Sta. Res. Bul. 111. 1928.
12. MITTER, J. H., and R. N. TANDON. A note on Sclerospora graminicola, (Sacc.) Schroet. in Allahabad. Jour. Indian Bot. Soc. **9**: 243. 1930.

TRANSMISSION OF SMUT AND MOLDS IN FIGS

H. N. Hansen and A. E. Davey

During the past 3 decades the fig growers of California have suffered increasing losses due to various rots, decays, and fermentations of their product caused by cryptogamic organisms carried into the cavities of the figs by various insects. As early as 1892 Newton B. Pierce (10) stated: ". . . . the growers had to contend with a destructive fermentation of the fruit which often caused the loss of nearly the entire crop." And further: "The fruit is inoculated by insects." Howard (8) also suggests insect transmission of souring, while Condit (3) and Hodgson (6) mention a similar possibility in the case of the fungus infection called smut. Condit (4) states that souring is only another name for fermentation caused by the action of yeasts, molds, and bacteria. He attributes the introduction of these organisms into figs to insects, particularly "small beetles" and occasionally the vinegar fly. Phillips, Smith, and Smith (9), working particularly with "fig smut" (*Aspergillus niger* van T.), came to the conclusion that *Carpophilus hemipterus* L. and *Drosophila ampelophila* Loew. were the principal carriers of the causal organism. Caldis (1) has shown that *Fusarium moniliforme* var. *fici* Cald., which causes a rot (endosepsis) in caprified figs, is transmitted from the capri to the edible fig by the caprifying insect *Blastophaga psenes* L. The same author (2) also shows that *Carpophilus hemipterus* L. carries yeasts into the interior of both caprified and parthenocarpic figs. From this it will be seen that insect transmission of fig-spoiling organisms has long been recognized.

In speaking of insect transmission in connection with fig spoilage it has, however, been generally assumed that, so far as the parthenocarpic varieties of figs were concerned, no insects entered them until they were nearly ripe and the eye well opened (1, 9). In 1929 the senior writer published a short note (5) in which he shows that thrips enter green figs long before the eye scales loosen and bring into them various cryptogamic organisms capable of producing spoilage. Later (11) predaceous mites were observed in overwintering capri figs (mamme) and found to be carrying various cryptogams.

"Smut and molds" is the designation of one of the spoilage grades in dried figs set up by Howard (7) of the United States Food, Drug and Insecticide Administration. This grade includes all figs showing visible fungus growths in the interior, whether of the black, dusty type of smut (*Aspergillus niger*) or the variously colored mycelial growths of other fungi. The present paper deals with the presence of mites[1] and thrips

[1] Unless otherwise specified, whenever mites are mentioned in this paper predaceous mites are meant.

in Adriatic (parthenocarpic) figs and their relation to the occurrence of smut and molds in these figs. Four places were selected near the fig-growing centers of the State (Madison, Modesto, Merced, and Fresno) from which representative samples of green, immature figs were obtained at intervals between July 1 and August 15, 1930 (Table 1). While the

TABLE 1.—*Showing seasonal variation of mites and thrips infestation in green Adriatic figs at Madison, California*

Date	Number of figs examined	Percentage of figs harboring mites	Percentage of figs harboring thrips	Percentage of figs harboring thrips or both
July 1	100	29.0	11.0	36.8
" 9	100	18.0	4.0	21.7
" 15	200	18.5	8.0	25.0
" 19	350	18.2	9.4	25.9
" 23	200	25.5	4.5	28.9
" 31	200	27.0	2.5	28.8
August 4	200	25.5	6.0	30.0
" 8	100	34.0	5.0	37.3
" 15	100	21.0	0.0	21.0
Totals	1,550	23.2	6.1	27.2

fig is very young up to about the size of a large hazelnut, the eye scales are quite pliable, but, as it develops further, the scales become hard and rigid and are able to offer considerable resistance to any insect trying to enter the fruit. Later, as the fruit matures, the eye scales again loosen and spread apart until at full maturity there may be a clear passage to the interior of the fig from 2 to 5 mm. in diameter. It should be borne in mind that the fig tree produces its main crop on current-season wood and that new fruits are formed over the comparatively long period of from 4 to 6 weeks, so that figs in all stages of maturity may be found simultaneously on the same tree. In collecting our samples only green figs were taken and these in numbers representative of the various maturity stages from the hazelnut size up to the time when the eye scales begin to loosen again. The figs were taken to the laboratory at Davis, split into halves, and examined for mite and insect infestation by means of dissecting microscopes. No attempts were made to determine the frequency of occurrence of individual species of mites and thrips or to determine numbers of mites and thrips in

TABLE 2.—*Extent of infestation of mites and thrips in green Adriatic figs*

Place	Number of figs examined	Figs showing predaceous mites		Figs showing thrips		Figs showing mites and thrips	
		Number	Per cent	Number	Per cent	Number	Per cent
Madison	1,550	359	23.2	95	6.1	422	27.2
Modesto	1,800	549	30.5	50	2.8	562	31.2
Merced	1,000	206	20.6	12	1.2	209	20.9
Fresno	920	268	29.1	24	2.6	282	30.6
Totals	5,270	1,382	26.2	181	3.4	1,475	28.0

individual figs. The data are summarized in table 2. During the progress of this examination mites and thrips taken from the interior of the figs were cultured on nutrient agar from time to time to determine the abundance and diversity of flora carried by them. (Table 3.)

TABLE 3.—*Results of culturing mites and thrips from the interior of green Adriatic figs*

	Number cultured	Number sterile	Percentage sterile	Number carrying cryptogams	Percentage carrying cryptogams
Mites	266	122	43.5	144	54.2
Thrips	194	78	40.2	116	59.8
Totals	460	200	43.5	260	56.5

In order to show the effect of maximum infestation of mites and thrips and, at the same time, exclude larger insects (mainly *Carpophilus hemipterus* and *Drosophila ampelophila*) from entering the figs, the following experiment was devised. During August 10 to 15 the still unopened eyes of 1,557 figs were effectively sealed by placing on the eye scales of each a

TABLE 4.—*Effect of sealing the eyes of green Adriatic figs on the occurrence of smut and molds*

Treatment	Number of figs examined	Figs with smut and molds	
		Number	Per cent
Sealed	1,557	359	16.6
Unsealed controls	400	54	13.5

small dab of tanglefoot preparation. Such treatment did not appear to injure the fruit in any way, as it developed and matured in normal manner and season. The treated figs were allowed to mature on the trees and were not collected until they had dropped to the ground, after which they were taken to the laboratory, split open, and examined for smut and molds. As control, 400 mature figs were picked from the ground under surrounding trees and examined likewise. (Table 4.)

DISCUSSION OF RESULTS

The percentages in table 1 show considerable variation of infestation of mites and thrips throughout the season but no considerable or consistent increase as the season advances. This indicates that the major part of infestation takes place while the fig is very young and the eye scales pliable.

Table 2 shows that mite and thrips infestation in green, immature figs is quite extensive and general throughout the State. It also shows considerable variation in amount of infestation between localities. Compare figures for Modesto and Merced. Annual variations (not shown in the tables) may, however, be much greater. For example, the table shows thrips infestation for Madison to be 6.1 per cent, whereas thrips infestation at the same place, the previous year, was over 18.0 per cent. The factors responsible for these variations are not evident.

Table 3 shows that more thrips than mites are carriers of cryptogams. This is probably due to the greater size of the insects. Since nearly half of the mites and thrips found in figs are sterile, direct prediction as to the amount of loss to be expected from smut and molds can not be made on the basis of the percentage of figs infested. However, an indicative figure may be obtained by multiplying the percentage of figs infested by that of mites and thrips carrying cryptogams. For example, the total percentages in tables 2 and 3 $\frac{28.0 \times 56.5}{100}$ gives 15.2 per cent, a close approximation to the percentage of actual smut and mold loss shown in table 4.

The data given in table 4 indicate strongly that the major part of smut and mold loss is due to cryptogamic organisms carried into the green figs by predaceous mites and thrips long before the eye scales begin to loosen, and they clearly show that the presence of *Carpophilus hemipterus* and *Drosophila ampelophila* is not at all necessary for the occurrence of this type of spoilage. The higher percentage of smut and molds in the figs with sealed eyes probably can be attributed to closing of the eye, which might be expected to create a more humid condition in the fig, favoring germination of mold spores.

The cryptogamic flora in green and ripe figs and on mites and thrips, cultured, included the following species named in the order of the fre-

quency of their occurrence: Miscellaneous fungi, bacteria, *Hormodendrum* spp., *Aspergillus* spp., *Penicillium* spp., *Alternaria* spp., *Rhizopus* spp., *Acrostalagmus* sp., and a few yeasts.

FAUNA

The mites found in green figs consisted of *Sejus pomi* Parrott, *Cheyletus,* sp., and two other unidentified species. The mites are very small, varying in length from 0.4 to 0.9 mm. They have been observed to feed on the common fig mite, *Eriophyes fici* Ewing. These predaceous mites usually confine their activity to the eye region of the fig and are seldom found deep in the interior. The common fig mite was found in about 90 per cent of all figs examined. It is apparently insignificant as a carrier of cryptogamic organisms but may be of considerable importance in attracting predaceous mites. Of thrips, the following were found: *Frankliniella tritici* (Fitch), *F. tritici* var. *californicus* (Moulton), *Thrips bremneri* (Moulton), *Liothrips ilex* (Moulton), *Heliothrips fasciatus* (Perg), and the predaceous thrips, *Leptothrips mali* (Fitch). All of the mites listed and at least 2 of the thrips, *F. tritici* and *Leptothrips mali,* have been found to breed within the figs of all crops, both edible and capri. These thrips, with the exception of *mali,* feed upon the tender floral parts of the fig. They are thus not confined to the eye region, as are the mites, but wander throughout the interior of the fig and, for this reason, they are probably more *effective* as carriers of spores.

SUMMARY

Some of the literature relating to transmission of fig-spoilage organisms is reviewed.

It is shown that figs are entered by mites and thrips long before the eye scales begin to loosen.

It is demonstrated that mites and thrips are carriers of cryptogamic organisms capable of producing smut and molds in figs.

It is proved experimentally that presence of the larger insects *Carpophilus hemipterus* and *Drosophila ampelophila* is not necessary for occurrence of smut and molds in figs.

The list of the mites and thrips and the fig-spoilage organisms they carry is appended.

LITERATURE CITED

1. CALDIS, P. D. A rot of the Smyrna fig in California. Science, n. s. **62**: 161–162. 1925.

2. ————————. Souring of figs by yeasts and the transmission of the disease by insects. Jour. Agr. Res. **40**: 1031–1051. 1930.

3. CONDIT, I. J. Proceedings of the Fig Institute **1917**: 1–111. Published by the Fresno Herald, Fresno, California. 1917.
4. ————————. Some causes of the souring and splitting of figs. Fig and Olive Jour. 3 (9): 13–16. 1919.
5. HANSEN, H. N. Thrips as carriers of fig-decaying organisms. Science, n. s. **69**: 356–357. 1929.
6. HODGSON, R. W. Black smut of figs. Calif. State Comm. Hort. Monthly Bul. 7: 188–189. 1918.
7. HOWARD, B. J. Fig testing. U. S. Dept. Agr., Food, Drug and Insecticide Administration. 11 pp. 1929. (Mimeographed).
8. HOWARD, L. O. Smyrna fig culture in the United States. U. S. Dept. Agr. Yearbook **1900**: 79–106. 1901.
10. PIERCE, N. B. Investigations of the special agent in California. U. S. Dept. Agr. Rpt. **1892**: 238–241. 1893.
11. SMITH, R. E., and H. N. HANSEN. Fruit spoilage diseases of figs. Calif. Agr. Exp. Sta. Bul. 506. 1931.

SEED DISSEMINATION IN FUSARIUM WILT OF PEA[1]

W. C. SNYDER[2]

INTRODUCTION

It has been only since the paper of Linford (5), in 1928, that Fusarium wilt of pea has been recognized as a major disease of the garden and canning pea, *Pisum sativum* L., in the pea-seed and canning areas of the United States. Within the past 3 or 4 years the disease, attributed by him to *F. orthoceras* App. and Wr. var. *pisi* Linford, has been found in at least 10 States extending from Washington to Maryland (6).[3] The sudden occurrence of wilt in widely separated pea-growing areas not previously known to harbor the disease, and other field evidence, have suggested the possibility of dissemination with the seed, although it is common knowledge that local dissemination occurs by means of diseased vines, implements, water, and other agencies of soil transfer from wilt-infested fields.

In Linford's[3] 1928 survey, wilt was found for the first time in Montana and Idaho, where the bulk of the pea seed for the United States is grown. It appears significant that in both States foci of infection included the trial grounds of large seed companies. A less extensive survey by J. C. Walker and the writer, in 1930, showed the practical limit of infestation in this area was centered in the trial grounds. In the case of both States there has been a free exchange of seed lots with other States where wilt is known to occur, which points to strong circumstantial evidence in favor of seed transmission.

An attempt, therefore, has been made during the past year to definitely ascertain whether the disease may, under certain conditions at least, be transferred to clean soil with the seed. Results so far obtained are discussed in the body of this report.

Other related vascular diseases have been shown to be seed-borne. Bolley (1) reported that flax wilt may be spread by way of the seed, spores of the fungus being lodged upon the surface of the seed with dust at the time of threshing. Elliott and Crawford (3) have presented both field and laboratory evidence on the seed-borne occurrence of tomato wilt. They were able to follow the progress of the fungus up the vascular bundles

[1] Published with the permission of the Director of the Wisconsin Agricultural Experiment Station.

[2] Grateful acknowledgment is made to Dr. J. C. Walker under whose guidance the work has been carried out.

[3] Linford, Maurice B. Pea diseases in the United States in 1928. U. S. Dept. Agr., Bur. Plant Indus., Plant Disease Rptr. Sup. 67. 1929. (Mimeographed.)

of the plant into the pedicels and fruit. Tomato fruits so infected were reported to have 3½ per cent of their seed infected after a 7-month period, a smaller percentage of which harbored the fungus within the seed coats. Elliott (2) was able also to isolate the cotton-wilt fungus from the seed of infested bolls where, again, it was recoverable both on the surface and in the seed coats. In the case of pea wilt, however, the invading fungus is generally limited to subterranean portions of the plant entering only the lowest internodes of the aboveground plant. Linford (5) states "in no case was the fungus found within two internodes of the lowest pod." He removed asceptical seed from pods of wilted plants, plating them in Petri dishes of agar, but failed to obtain any trace of the parasite. These observations indicated that dissemination of wilt with pea seed occurs very rarely, if at all, and check with field experience where only an occasional wilt spot may be seen to occur in a several-acre field at the time disease is first observed to be present.

METHODS

In the summer of 1929 vines of susceptible varieties of pea, growing on wilt-infested soil, were collected in cases where some seed had been set before wilting was completed. Each internode of these plants from the ground upward was cultured for the wilt fungus and its position in relation to pod nodes carefully noted. In every case save one Linford's experience was duplicated. In the single exception out of about 50 plants the fungus was isolated from the stem internode immediately below the attachment of the lowest pod. The extreme limit of invasion, however, was confined, even in this case, only to the stem, for platings of both pedicel and seed of the lowest pod failed to give any trace of the pathogen.

A different approach was attempted the following year in order to test large populations of seed. The plan consisted in harvesting in bulk the seed occasionally set in the field by wilted plants, and sowing them in successive series upon wilt-free soil in the greenhouse. By this means the accumulation in the soil of small amounts of inoculum could be followed to the point of disease inception. Steamed soil was not used because of its inhibiting effect on early establishment of the fungus. The procedure was as follows:

A bench $3\frac{1}{2} \times 12$ ft. was selected in a section of greenhouse that had not previously been used for peas and was away from the main aisle. The bench was thoroughly washed, then repeatedly drenched with 1 to 400 hot corrosive sublimate. Virgin soil, far removed from cultivated fields, was obtained from beneath the turf on a knoll in Madison. There is every reason to believe it was free of the wilt organism, similar soil having been used repeatedly for healthy controls in inoculation work during the past

year with never a single case of wilt. No implement, conveyor, or article touched the soil from the time of its removal until its spreading in the bench, unless it was sterilized and every precaution was taken in planting, watering, and manipulating the bed to prevent any chance contamination with wilt. The opportunity for this was remote under these circumstances, especially since the organism bears few conidia. In this bed of soil was planted seed harvested from diseased plants collected in the summer of 1930.

The seed used was obtained from two different wilt fields in Wisconsin, one at Columbus, where the wilted plants in a 50 per cent resistant Alaska stock were pulled and the seed threshed, and the other at Beaver Dam, from small plots of Ashford and Acme (both completely susceptible varieties), which had been planted for a canning test but were lost through Fusarium wilt.

Beginning with September of 1930, 4 successive plantings were made using the seed described above, seeding the first 2 times with the Alaska seed, and the third and fourth times with a mixture of the Ashford and Acme seed, as indicated in table 1. The seed was thickly planted both in order to use large numbers of seed and because of a high percentage of nonviable, shriveled peas. This brought the total seed population planted in the bed from diseased plants to 7,900. A fifth planting of seed of the susceptible variety Perfection was sown at the conclusion of the tests. This last lot of seed was produced on wilt-free soil.

Each planting was allowed to remain in the bed for a period of 7 weeks, and a soil temperature was maintained within the optimum range for wilt development between 18° and 24° C. Under these conditions, on naturally infested soil, wilting normally occurs within 3 to 5 weeks. Upon the appearance of disease symptoms plants were pulled and cultured for positive identification of the pathogen. At the end of each series all plants were pulled and examined macroscopically.

RESULTS

In the first 3 crops, as shown by table 1, no symptom of wilt appeared. In the fourth 4 separate plants at different points on the bed showed distinct symptoms of wilt, both aerially and subterraneously and, on being cultured, each yielded colonies of the pea-wilt Fusarium. A pathogenicity test of these cultures, using Perfection seed upon artificially inoculated soil, proved their authenticity in each case.

From the fifth seeding with Perfection harvested from a wilt-free area, a few suspicious plants were cultured before the plants dried up from extreme heat. Of these, 3 were found to be infected and *Fusarium orthoceras* var. *pisi* was recovered from them and used successfully in further patho-

genicity tests. It is probable others might have been found had this run gone to completion, but the 3 obtained suffice to demonstrate clearly that the wilt fungus had become established in the soil.

TABLE 1.—*Appearance of wilt in successive plantings of seed from pea plants infested with Fusarium orthoceras* var. *pisi*

Series	Variety	Source of seed	Number of seeds planted	Number of wilted plants
1	Alaska	Columbus, Wisconsin	1,850	0
2	"	" "	1,850	0
3	{ Ashford { Acme	Beaver Dam, Wisconsin	2,100	0
4	"	" " "	2,100	4
5	Perfection[a]	Idaho	2,000	3[b]

[a] The seed used in the fifth series was grown on wilt-free soil.

[b] Incomplete reading owing to death of the plants from extremely high temperatures.

DISCUSSION

These results, supported by field observations of both Linford and the writer, taken over 2 periods of years, are interpreted as definite evidence that the Fusarium wilt of pea may be occasionally, though not abundantly, carried with the seed. They do not reveal the manner in which the fungus is carried, whether within the seed coats or upon the surface as spores adhering to the seed coats in dust or associated with soil particles and other bits of refuse attending the seed.

The conditions of these trials should be taken as a greatly exaggerated field condition in that only seed from wilted plants was used. Yet, out of a population of about 8,000 seeds, selected for their greater possibility of carrying infection, only 4 foci of infection were established in the seed bed. These first 4 cases of wilt may be interpreted as the accumulative effect of small amounts of inoculum introduced with the various series and not necessarily the result of inoculum brought in with only the fourth seeding.

It is common observation that even in factory-cleaned seed, an occasional pea seed, especially of the wrinkled types, may be found with a small piece of soil lodged in an indentation of the surface. Since the fungus produces chlamydospores abundantly, it is plausible to conceive of the fungus being carried in this medium. In addition, since the fungus is occasionally found in the stem node giving origin to the lowest pod of a wilted plant, it is likely that once in a great while invasion of the pedicel and thence

of a seed may take place. Invasion of the seed may also occur in an additional manner where the pods lie near or upon the ground. Jones (4) in testing pea seed for the presence of *Ascochyta* spp. isolated from surface-sterilized seed various fungi including *Fusarium* spp. as well as *Ascochyta* spp. and bacteria. Since other *Fusarium* spp. are shown to be present occasionally in the interior of pea seed, there seems to be no reason why *F. orthoceras* var. *pisi* should not also enter in this manner, since it grows as a saprophyte on the plant refuse and may well be expected to follow active pod invaders or even directly enter maturing pods in contact with the soil.

The establishment of seed transmissibility of the Fusarium-wilt organism is of little concern from the practical standpoint in regions where wilt is already present, but it does confirm a long-held opinion of those in contact with the wilt problem. Seed treatment on a commercial scale to catch the rarely infected seeds would be impractical and uneconomical and with the increasing use of wilt-resistant varieties (6) in the important wilt areas, it will be of even less concern. However, it is clear that pea seed from wilt fields is a potential source of primary inoculum for any wilt-free soil suited to the establishment of the fungus.

SUMMARY

The organism causing the Fusarium wilt of pea is shown to be occasionally carried with pea seed harvested from wilt-infested land. This means of transmission, while relatively rare, is, nevertheless, to be regarded as a potential source of primary inoculum whereby the organism may be introduced into non-infested soil.

The exact manner in which the fungus is carried is not known.

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LITERATURE CITED

1. BOLLEY, H. L. Flax and flax seed selection. N. Dak. Agr. Exp. Sta. Bul. **55**: 171–198. 1903.
2. ELLIOTT, J. A. Cotton-wilt, a seed-borne disease. Jour. Agr. Res. **23**: 387–393. 1923.
3. ———————, and R. F. CRAWFORD. The spread of tomato wilt by infected seed. Phytopath. **12**: 428–434. 1922.
4. JONES, LEON K. Studies of the nature and control of blight, leaf and pod spot, and foot rot of peas caused by species of Ascochyta. N. Y. (Geneva) Agr. Exp. Sta. Bul. 547. 1927.
5. LINFORD, MAURICE B. A Fusarium wilt of peas in Wisconsin. Wis. Agr. Exp. Sta. Res. Bul. 85. 1928.
6. WALKER, J. C. Resistance to Fusarium wilt in garden, canning and field peas. Wis. Agr. Exp. Sta. Res. Bul. 107. 1931.

EFFECT OF BUNT ON HEIGHT OF WHEAT PLANTS[1]

E. N. Bressman

Bunt, or stinking smut, of wheat includes two species, *Tilletia tritici* (Bjerk.) Winter and *Tilletia levis* Kühn. *Tilletia tritici* is frequently referred to as "low smut" and *T. levis* is called "high smut," because of the height of the bunted culms in comparison with normal ones. In 1918 Potter and Coons[2] confirmed the earlier observations of a Michigan farmer that there were two types of infected plants, "high" and "low."

Because of the common belief that the species differ in the above way, record was made of the relative heights of the diseased and normal plants in the writer's 1928 tests at both Moro and Corvallis, Oregon, conducted to determine physiologic forms of bunt. There were 430 rows from 8 to 10 ft. in length at each place. The seed of 10 varieties of wheat, Hybrid 128, Albit, Ridit, Oro, Turkey × Bearded Minnesota 48, Hussar, Regal, Martin, White Odessa, and Banner Berkeley, was inoculated with 43 collections of bunt spores. Details of the procedure and the sources of these collections are published elsewhere.[3]

At the time of making smut counts notes were taken on the relative height of the bunted plants as compared with noninfected plants. The results are tabulated in table 1. The estimates at Moro, Oregon, were made by J. Foster Martin.

A summary of the classification of the height of bunted plants shows that of the 387 rows of *Tilletia levis*, 293 were classified as low and 94 were called normal or low to normal, which is a ratio of about 3 to 1. Of the 223 rows of *T. tritici*, 194 were classified as low or low to normal and 29 normal, which is a ratio of 6.7 to 1. The *T. levis* classification on a 6.7 to 1 basis gives a value of 25.67 for X^2. This means that the odds are several thousand to one that the difference in height of smutted culms thrown by these two species is not due to chance alone.

On a 3 : 1 basis the 6.7 : 1 ratio has a P. E. of 5.2. The deviation is 26.75 and deviation divided by P. E. is 5.1 or odds of more than 1,000 to 1 that the two species are different in height of smutted culm.[4] Both

[1] Published as Technical Paper No. 155 with the approval of the Director of the Oregon Agricultural Experiment Station, a contribution of the Department of Farm Crops.

[2] Potter, A. A., and G. W. Coons. Differences between the species of the Tilletia on wheat. Phytopath. 8: 106–113. 1918.

[3] Bressman, E. N. Varietal resistance, physiologic specialization, and inheritance studies in bunt of wheat. Oreg. Agr. Exp. Sta. Bul. 281. 1931.

[4] Hayes, H. K., and R. J. Garber. Breeding crop plants. 438 pp. 2nd Ed. McGraw-Hill, New York. 1927.

TABLE 1.—*Relative height of bunted heads in 10 varieties of wheat, inoculated with 43 collections of bunt. T—Tilletia tritici and L—Tilletia levis. Corvallis (C) and Moro (M), Oregon, 1928*

Variety	1a[a] – L.		1b – L.		1c – L.		1d – T.		1e – T.		2 – L.		3 – T.		4 – L.		5 – T.		6a – T.		6b – T.	
	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M
Oro	L[b]	N	–	L	L	L	L	L	L	L	–	L	–	L	L	L	–	L	L	L	–	L
Turk. × B.M. 48	–	N	–	L	–	L	L	L	–	L	L	L	L	L	–	L	–	L	–	L	–	–
Regal	–	L	–	L	L	L	L	N	L	L	L	L	L	L	L	L	L	L	–	–	–	L
Ridit	L	L	–	L	L	L	L	L	–	–	L	L	L	L	L	L	L	–	–	L	–	L
Hybrid 128	N	L	N	L	N	LN	L	L	N	L	N	L	N	L	N	L	L	L	L	L	L	L
Albit	L	L	L	N	L	N	L	L	LN	N	L	L	L	L	L	N	–	–	–	L	–	–
Hussar	–	–	–	L	L	L	–	L	–	L	–	–	N	L	L	L	–	L	–	–	–	–
Martin	N	L	N	L	L	L	N	L	L	L	–	L	L	L	N	L	–	–	N	L	N	–
Banner Berkeley	–	–	L	L	LN	L	L	L	L	L	L	–	L	L	L	L	L	L	–	–	–	–
White Odessa	–	–	N	L	L	L	LN	L	L	L	–	–	L	L	L	L	–	L	–	L	–	–
Average	8L	4N	11L	4N	15L	4N	17L	2N	14L	2N	12L	1N	17L	2N	16L	2N	11L	0N	9L	1N	5L	1N

Variety	7a – T.		7b – T.		7e – T.		8a – T.		8b – L.		8e – T.		9 – T.		10 – T.		11 – L.		12a – T.		12b – T.	
	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M
Oro	–	L	–	L	–	–	L	L	N	L	–	L	L	L	–	L	–	L	–	L	L	–
Turk. × B.M. 48	N	L	–	–	–	–	–	L	–	L	–	L	N	L	L	L	L	L	–	L	–	–
Regal	L	L	–	L	–	L	–	L	L	L	L	L	L	L	–	L	L	L	–	L	–	–
Ridit	–	–	–	L	–	–	–	L	–	L	L	–	L	–	–	–	–	–	–	–	L	L
Hybrid 128	N	LN	N	L	L	L	N	LN	N	L	L	L	L	L	N	N	N	L	N	LN	N	LN
Albit	L	LN	L	–	L	L	L	L	L	N	L	L	N	N	N	L	L	L	–	L	–	–
Hussar	–	L	–	–	–	–	–	L	–	L	–	L	LN	L	–	L	–	L	–	–	–	–
Martin	–	L	N	L	L	L	–	L	L	L	L	L	L	L	L	L	L	L	L	L	L	L
Banner Berkeley	L	L	L	L	L	–	L	L	L	L	–	L	L	L	L	–	L	–	–	–	–	–
White Odessa	L	L	–	L	L	L	L	L	LN	L	–	L	N	L	L	L	L	L	–	–	–	–
Average	13L	2N	9L	2N	10L	0N	14L	1N	13L	4N	14L	0N	15L	4N	11L	3N	14L	1N	7L	1N	6L	1N

[a] 1a, 1b, etc., are collections of bunt.

[b] L is lower than normal culm. N is same height as nomal culm. LN varies from lower to same height as normal culm. These are classified as L or N, depending upon species. Where no culm height is indicated, no bunt occurred.

[a] 1a, 1b, etc., are collections of bunt.
[b] L is lower than normal culm. N is same height as nomal culm. LN varies from lower to same height as normal culm. These are classified as L or N, depending upon species. Where no culm height is indicated, no bunt occurred.

TABLE 1.—*Relative height of bunted heads in 10 varieties of wheat, inoculated with 43 collections of bunt. T—Tilletia tritici and L—Tilletia levis. Corvallis* (C) *and Moro* (M), *Oregon, 1928*

Variety	13 – L.		14 – L.		15 – L.		16a – L.		16b – L.		17 – L.		18 – L.		19a – L.		19b – T.		19c – L.		19d – T.	
	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M
Oro	N	LN	L	LN	L	L	L	L	L	L	L	L	L	L	–	L	L	L	L	L	–	L
Turk. × B.M. 48	–	L	N	L	–	L	L	L	L	L	L	L	L	L	–	L	N	L	L	L	L	L
Regal	L	L	–	L	L	L	L	L	–	L	L	L	L	L	L	LN	N	L	L	LN	L	LN
Ridit	L	L	L	L	L	–	L	L	–	L	–	L	L	L	L	–	–	L	–	L	–	–
Hybrid 128	N	L	N	L	N	LN	N	L	N	N	N	LN	N	LN	N	LN	N	LN	N	L	N	LN
Albit	L	N	–	L	L	L	–	L	–	L	L	LN	–	L	L	LN	–	L	N	L	N	LN
Hussar	–	L	–	L	–	L	–	–	–	–	–	–	–	–	–	L	–	L	L	LN	–	L
Martin	L	L	L	L	–	L	L	–	N	L	L	L	L	L	N	LN	L	L	L	L	N	LN
Banner Berkeley	–	L	–	–	L	L	–	–	–	–	–	L	L	L	L	L	–	–	L	L	L	LN
White Odessa	L	L	–	–	L	L	–	–	–	–	–	L	L	L	N	L	–	–	N	L	N	L
Average	13L	4N	10L	3N	14L	2N	11L	1N	8L	3N	12L	3N	15L	2N	9L	7N	10L	3N	14L	5N	12L	4N

Variety	19e – L.		20 – L.		21 – L.		22 – L.		23 – L.		24 – L.		25 – L.		26 – L.		27 – L.		28 – L.	
	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M	C	M
Oro	–	L	–	L	–	L	–	L	L	L	L	L	N	L	N	L	L	LN	N	LN
Turk × B.M. 48	–	–	L	L	N	L	–	L	–	L	N	L	L	L	L	L	L	L	L	LN
Regal	–	L	–	L	N	L	–	L	L	LN	L	L	L	L	N	L	L	LN	N	LN
Ridit	–	–	–	L	–	L	–	–	–	–	–	–	L	–	L	–	–	L	L	L
Hybrid 128	N	LN	N	LN	N	LN	N	L	N	LN	N	L	N	L	N	LN	N	LN	N	LN
Albit	–	LN	L	LN	–	LN	–	LN	L	LN	L	LN	L	L	–	LN	L	LN	L	LN
Hussar	–	L	–	L	–	L	–	–	–	L	–	L	–	–	–	L	–	L	–	L
Martin	L	L	L	L	L	L	L	L	L	L	L	L	L	L	L	L	N	L	L	L
Banner Berkeley	–	–	–	–	–	–	–	–	L	L	L	L	–	–	L	L	L	L	L	L
White Odessa	–	–	–	L	–	L	–	–	N	L	N	L	L	–	–	L	L	L	L	L
Average	5L	3N	10L	3N	8L	5N	6L	2N	10L	6N	13L	4N	12L	2N	11L	5N	12L	6N	11L	8N

[a] 1a, 1b, etc., are collections of bunt.
[b] L is lower than normal culm. N is same height as nomal culm. LN varies from lower to same height as normal culm. These are classified as L or N, depending upon species. Where no culm height is indicated, no bunt occurred.

species tend to produce plants lower than the normal. But, the foregoing analysis shows that, on the average, the *Tilletia levis* infected plants are taller than those infected with *T. tritici*. If the rows with smutted plants of varied height were not considered, the difference would not be so great.

Hybrid 128 produced taller and more varied infected plants than did the other varieties in the trial. Of the 52 *T. levis* infected Hybrid 128 rows, 13 were classified as low and 39 as normal or low to normal. Of the 34 *T. tritici* infected Hybrid 128 rows, 25 were classified as low or low to normal and 9 as normal. On the other hand, practically all of the smutted Banner Berkeley plants were low.

Collection *1e* is of special interest, as it comprised several heads of *Tilletia tritici* produced on plants that were as tall as those of nonsmutted plants. It was collected at Corvallis from what has come to be known as "Palouse" bunt. This collection was obtained in the fall of 1926 from the office of Federal Grain Supervision, Bureau of Agricultural Economics, United States Department of Agriculture, Oregon. The original material is the same as that used by D. E. Stephens at Moro, Oregon, and mentioned by Gaines[5] as showing striking results at Moro. Collection *1e* did not continue to show these tall bunted plants in 1927–28. This indicates that environmental conditions, as well as the species, may affect the height of the smutted plants. Also, the results with this collection may account for the variation in height of smutted plants found in certain rows. It is of interest to note that both collections of *1d* and *1e* are classified by the writer as physiologic form 7 because they are similar in their smutting of the differential hosts. Collection *1d* consisted of the low infected plants and *1e* the high infected plants found in Palouse bunt.

In general, *Tilletia levis* produces a taller infected plant than *T. tritici*, but it is not a constant characteristic of the species. Varieties and environmental conditions also have an effect on the height of the infected plants. There appear to be other differences of greater importance within the species than between the two.

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[5] Gaines, E. F. New physiologic forms of *Tilletia levis* and *T. tritici*. Phytopath. 18: 579–588. 1928.

PHYTOPATHOLOGICAL NOTES

An atypical lesion on cotton leaves caused by Bacterium malvacearum.—In the summer of 1929 the writer received from O. C. Boyd some cotton leaves collected in North Carolina, with a query as to the cause of the large dead areas. He reported that this type of lesion was causing more serious defoliation than the usual angular spot.

The spots, marginal or otherwise, 1 to 3 cm. wide, irregular in shape, appeared to be the result of vascular rather than stomatal infection, formed by a gradual and progressive fading out and dying of the tissues; when marginal, advancing from the margin inward. In the pale green or gray areas the veins were darkened, not with the well-known external blackening occurring in typical angular spot, but with such as results from an internal disturbance (Fig. 1).

Sections cut across such lesions show a heavy infection in the vascular system. Bacteria pour out in great numbers from the cut ends of the veins but not from the intervening tissue. This is not true of typical angular spot, from which the bacteria are extruded from all of the tissues of the entire area of the spot. Bacteria were found in the veins at some distance from the discolored area and in one instance were plated from the petiole. In no case, however, was it found that the bacteria had entered through the petiole. They had spread from an infection on the blade.

The point of entry, whether by a water pore or through an inconspicuous lesion on a vein, as sometimes appears to be the case, has not been determined. It seems evident, however, that this strain of the organism is able under some conditions to enter and spread through the vascular system.

Plates were poured and a yellow bacterial organism obtained that resembles *Bacterium malvacearum*. Inoculations made by spraying cotton leaves in the hothouse, using subcultures from these isolations, gave typical angular-spot lesions on leaf blades, petioles, veins, and bolls, as well as large, pale spots like those from which the culture used was obtained. Reisolations and repeated inoculations with reisolations have given the same results.

Parallel inoculations with a freshly isolated strain of *Bacterium malvacearum* from typical angular spot from another source gave the typical angular lesions but none of the systemic infections. No record has been found of the above type of spot, nor has the writer ever obtained it before in numerous greenhouse inoculations made in the past with *Bact. malvacearum* from various sources. Since only minor cultural differences have been found between this strain and strains from typical lesions, it seems

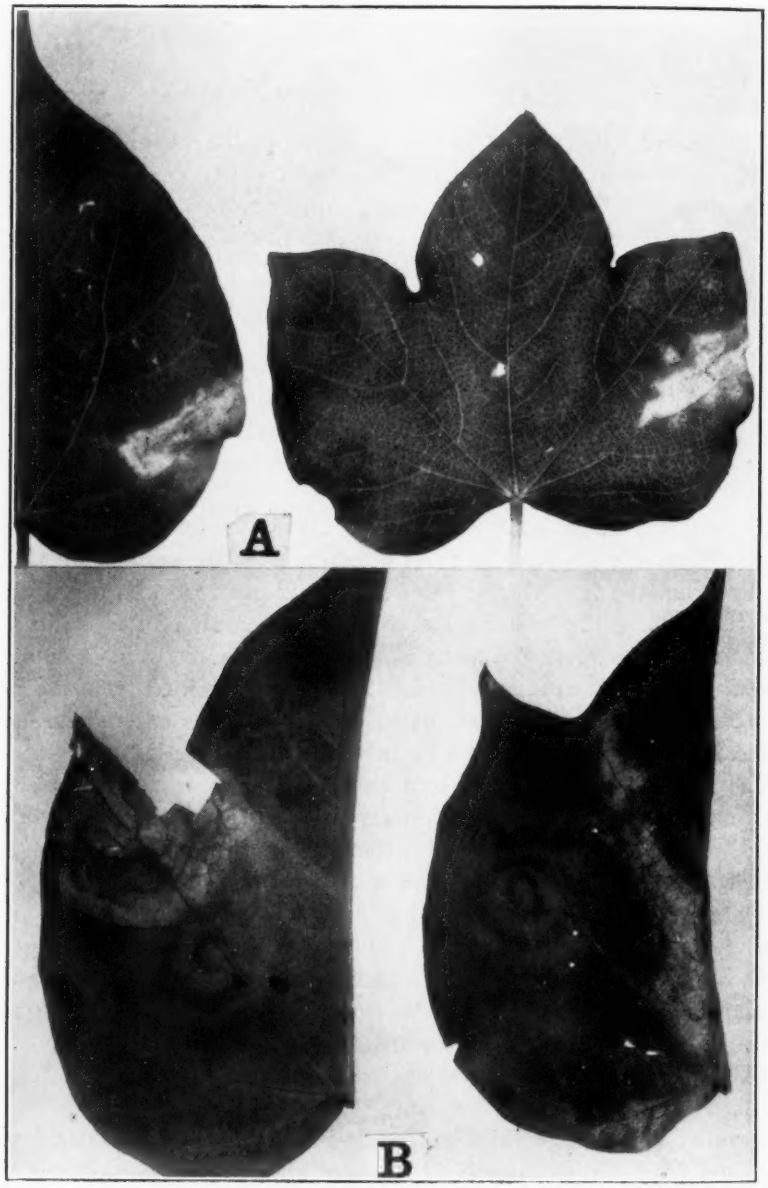

FIG. 1. A. Young spots produced by inoculation with the North Carolina strain of *Bacterium malvacearum*. B. More advanced stage, showing darkened veins.

evident that the organism in question is *Bact. malvacearum*.—MARY K. BRYAN, Bureau of Plant Industry, United States Department of Agriculture.

Root, crown, and shoot rot of milo.—In the Southwestern States, where grain sorghums are grown extensively, milos have, in some localities, been showing a marked susceptibility to a certain root, crown, and shoot rot. On certain field experiment stations the disease has appeared first on plots continuously cropped to milo for a number of years or on plots where there have been relatively short intervals between crops of milo. In the more severe cases it has caused complete failure of the crop and is becoming a limiting factor in milo production.

Dwarfing of the plants and lack of heading are common symptoms of the disease. (Fig. 1.) Where the disease is complicated by chinch-bug in-

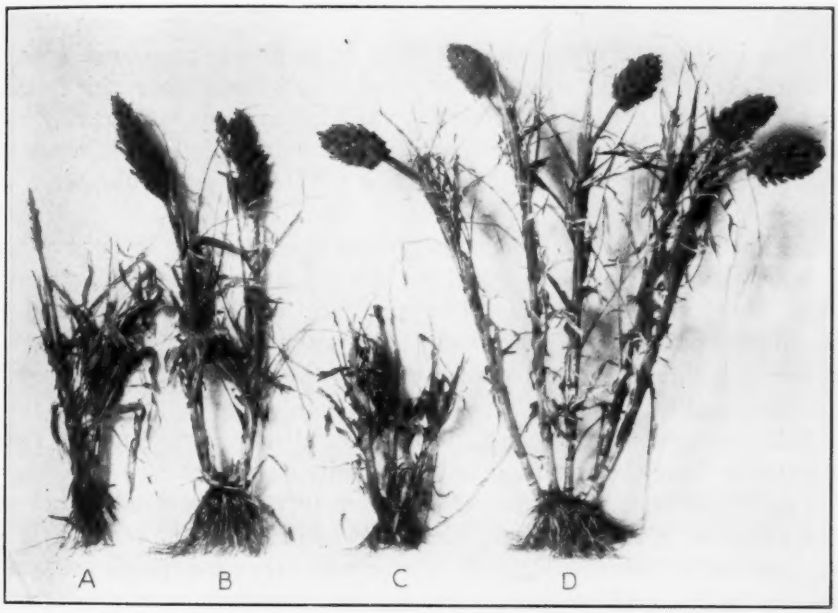

FIG. 1. A, Diseased, and B, healthy plants of Custer milo; and C, diseased, and D, healthy plants of Dwarf Yellow milo. Garden City, Kansas, 1930.

jury, there is an abnormal amount of tillering. Where there are no chinch bugs, plants may grow 10 to 12 in. in height before any symptoms of the disease appear. The lowest leaves turn yellow along the margins, the yellow color gradually spreading over the entire leaf and appearing on the leaves above. The plants remain stunted and produce no heads, or only small ones, and a gradual drying and decay of the plants follow. These symptoms above ground are usually accompanied by a dark red discoloration of the central cylinder of the roots and of the interior of the crown. This red discoloration may spread throughout the crown and up into the

lower nodes of the stalk and is often followed by a brown discoloration and decay of roots and crown. In some cases there is decay of the central shoot with or without the other symptoms.

These symptoms have occurred under different soil and climatic conditions of southeastern, south-central, and northwestern Texas, northwestern Oklahoma, northeastern New Mexico, western Kansas, and southeastern California.

The following facts point to the parasitic nature of the disease: Both susceptible and resistant varieties of sorghum have shown few or no disease symptoms when grown in soil where the disease has not been previously evident. Susceptible varieties grown in soil that had produced the disease previously have shown 100 per cent diseased plants, while resistant varieties in the same soil have shown few or no disease symptoms. When such soil is sterilized by steam under pressure or by treatment with formaldehyde, both susceptible and resistant varieties have grown normally and have remained healthy. Preliminary tests have indicated that the disease symptoms may be produced by the addition of infested soil or diseased roots to the sterilized soil.

While all of the western soils tested have been found to be more or less alkaline, the most alkaline, tested by the quinhydrone method, was pH 7.41.[1] By the LaMotte method the reading was pH 8.0, both for the clean as well as the disease-producing soil. Analysis[1] of the infested soil showed no excess of total soluble salts such as might be concerned in the production of disease symptoms.

Soil treatments with potash, phosphates, nitrates, and complete fertilizers so far have shown no indications of soil deficiencies.

Greenhouse tests with infested soils from three different localities have shown that the plants react somewhat differently in the different soils, and it is possible that different factors may be responsible for the disease symptoms in the different localities.

A white bacterium and strains of Fusaria have been consistently isolated from diseased plants from different localities, but, to date, pure-culture inoculations have not demonstrated the pathogenicity of any of these organisms.

Varietal tests conducted at Garden City, Kansas, have shown that all typical milo varieties are very susceptible to the disease in question and that most milo hybrids are more or less susceptible. Kafirs, feteritas, sorgos, and Fargo Straightneck milo (a kafir-milo hybrid) are resistant. Studies are being conducted cooperatively by the Division of Cereal Crops and Diseases, Bureau of Plant Industry, the Kansas Agricultural Experi-

[1] Electrometric pH determinations and soil analyses were made by the Bureau of Chemistry and Soils, United States Department of Agriculture.

ment Station, and the Texas Agricultural Experiment Station.—CHARLOTTE ELLIOTT, Bureau of Plant Industry, United States Department of Agriculture; F. A. WAGNER, Garden City Branch of the Kansas Agricultural Experiment Station, Garden City, Kansas; and L. E. MELCHERS, Kansas Agricultural Experiment Station, Manhattan, Kansas.

Sulphur dioxide injury of tomatoes.—For several years occasional cars of tomatoes showing deeply sunken areas about the stem end have been observed on the markets. The tomatoes in most cars appeared normal in every respect except for the sunken stem end and a bleached greenish tan discoloration of the immediately surrounding tissues. (Fig. 1). Prelimi-

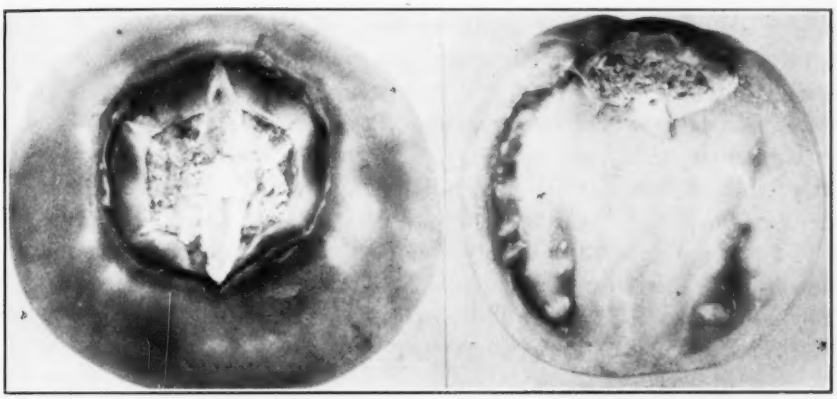

FIG. 1. Stem-end and longitudinal sectional views of a tomato showing sunken cracked areas and bleached surrounding tissues.

nary investigations indicated that the injury was not due to a parasite but was possibly induced by some unfavorable environmental condition.

The similarity of this injury to that caused by fumigation of watermelons with formaldehyde[1] suggested the possibility that the tomatoes were affected by some gas. Laboratory experiments demonstrated that this type of injury could be produced by exposing tomatoes in a closed container to the fumes from burning sulphur. Later investigations of certain cars of tomatoes showing the symptoms under consideration established the fact that the fruits had been subjected to fumigation by burning sulphur in the loaded cars at shipping point.

Tomatoes injured by exposure to sulphur dioxide gas have been observed on the markets in stock from California and Florida. Although only a few cars have been found showing this type of trouble, the serious-

[1] Ramsey, G. B. Fumigation injury of watermelons. Phytopath. 25: 479–481. 1925.

ness of the injury warrants mention. It may result from burning sulphur in cars of tomatoes or using sulphur dioxide gas, as is practiced with certain commodities in California, to reduce the amount of decay during transit and marketing. However, it appears that no experimental data are available to show that this treatment is of value in reducing decay in cars of tomatoes.

The affected tomatoes show a very pronounced sinking and drying out of the tissues at the stem scar. The gas enters the fruits through this scar or through wounds elsewhere on the surface and the cells immediately surrounding the openings are killed and lose their water rapidly by evaporation, causing collapse of the affected regions. In some instances the stem scar is sunken ½ inch or more and the tissues around the scar are bleached a sickly greenish tan color, while the remainder of the tomato retains the normal color except where mechanical injury is evident. Wherever the cuticle and epidermis of a fruit show a wound, the surrounding tissues are sunken and bleached, like those around the stem end. These sunken areas dry and crack, thus opening the way for various secondary decay-producing organisms, such as Alternaria, Rhizopus, and Fusarium. Decay induced by these fungi becomes especially damaging in the greener fruit that is held under the humid and high-temperature conditions of the ripening rooms on the receiving markets.—G. B. RAMSEY, Division of Horticultural Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture.

Black-walnut canker caused by a Nectria.[1] In a former issue of Science, Orton[2] reported the occurrence of a canker of black walnut, *Juglans nigra* L., in West Virginia. A survey of the State, begun early in the spring of 1930, has traced this disease in 24 counties extending from the northern to the southern boundary line of the State. The canker has been located also in Virginia and Pennsylvania and reported from Tennessee, Rhode Island, Wisconsin, and Ontario.

The frequent association of the red perithecia of a Nectria with the canker led to the assumption that this organism might be the causal agent. Consequently, series of inoculations were made at intervals of 2 weeks, beginning in January and ending in May.

All inoculations were made outdoors on the limbs and branches of older trees, as well as on the trunks of young ones, 2 to 5 years old. Conidia from pure cultures obtained from ascospores of the Nectria were used for

[1] Published with the approval of the Director, West Virginia Agricultural Experiment Station, Scientific Paper No. 99. The support of a David Allan Burt Fellowship in West Virginia University is also acknowledged.

[2] Orton, C. R. Black walnut canker. Science, n. s. 72: 142–143. 1930.

inoculum. The inoculations were made by puncturing the bark with the point of a sterile scalpel and inserting the inoculum in the wound, which was then wrapped with cotton and adhesive tape. Check wounds were treated in the same manner, except that no inoculum was inserted.

Of the 104 inoculations made between February 18 and April 28, inclusive, 82, or 79 per cent, showed a sunken area corresponding to the early stages of the canker. The results of the inoculations made in January could not be determined with certainty. This may have been due to the fact that the inoculations were made on large limbs on which the early stages of the disease do not show so readily, or that the temperature at the time the inoculations were made was too low for successful infection (−5° C.).

Of the 58 checks, 53, or 92 per cent, were undoubtedly free of infection. The other 5 showed a slight necrotic area that either was a local irritation or was caused by a natural infection occurring too late in the season to have assumed any size.

The fungus has been repeatedly reisolated from these artificially induced cankers.

The most noticeable character of the infections at this early stage is the sunken appearance of the dead area. This condition is due to the fact that, with the resumption of cambial activity, a new layer of wood is formed around the dead area by the noninfected cambium. The infection spreads more rapidly in the direction of the long axis of the stem, thus appearing more or less spindle-shape in outline. On 1- or 2-year old branches, which are normally light brown, the necrotic tissue may be further marked externally by a change to a dark brown. Scattered, tiny, whitish pustules protrude through the dead tissue. These consist of the sporodochia of the fungus. They appear most frequently at the lenticels, since these form the most natural breaks in the dead bark. Observed under the microscope, the sporodochium is seen to consist of a cushion-like stroma, from the outer, rounded surface of which the cylindrical conidia radiate in great numbers.—J. M. ASHCROFT, Department of Plant Pathology, Agricultural Experiment Station, Morgantown, W. Va.

Botrytis stem infection in pears. In a study of Botrytis rot of pears on fruit stored at Hood River, Oregon, it was found that certain lots of the 1930 crop showed a very high percentage of stem infection. The use of chemically treated paper wrappers to prevent the spread of Botrytis rot has made it easy to determine the number and place of initial infections.

Stem infection is characterized by the blackened tip of the stem having a definite line of demarcation between the healthy and the sound tissue. The diseased portion of the stem is usually soft and water-soaked and readily crumbles when rubbed between the thumb and finger. As the

storage season advances the discoloration moves down the stem and finally the fungus reaches and invades the fruit. It is unwise to try to hold fruit having infected stems until the late market, for the infection spreads from one fruit to another so rapidly that several fruits soon become rotten at each locus of infection.

The stem-infection type of rot was very sporadic on the 1930 crop in Hood River Valley. The fruit from most of the orchards showed very little of it, but that from certain orchards showed such severe stem infection that repacking was necessary because of rots resulting from the stem infection. Some boxes from the severely infected lots showed as high as 18 per cent stem infections. The lots showing severe stem infection came from orchards bearing a heavy cover crop of vetch or clover. Since dead and dying stems and leaves of such cover crops are known to be a favorable substrate for the growth and sporulation of Botrytis, an abundance of air-borne spores is probably present at pear-harvest time and available for inoculation of the abscissed stem ends of freshly picked pears.—J. S. COOLEY, Bureau of Plant Industry, United States Department of Agriculture, Washington, D. C.

Black rot of stocks. In the spring of 1929 and, again, in the spring of 1930 a wilt was prevalent among stock plants (*Matthiola incana* R. Br. var. *annua* Voss) in floral greenhouses in the vicinity of Knoxville, Tennessee. A bacterium was isolated from affected plants and its pathogenicity was proved by inoculations in wounds. In its morphology and physiology the organism resembled *Bacterium campestre* (Pam.) E. F. Smith, the pathogen causing black rot of crucifers. Moreover, the symptoms of the disease in stocks were similar to those produced by the black-rot organism in other crucifers.

Von Faber reported *Bacterium campestre* as causing black rot of *Matthiola incana* R. Br. in Germany in 1907[1]. From von Faber's paper Elliott, in her recent book, lists *M. incana* as a host of *Bact. campestre*.[2] Being unable to find any other report of black rot of stocks in the literature, the writer considers this probably the first record of *Bact. campestre* on Matthiola species in the United States.—L. M. COOLEY, Department of Botany, University of Tennessee, Knoxville, Tennessee.

[1] von Faber, F. C. Über eine Bakterienkrankheit der Levkoyen. Arb. K. Biol. Anst. Land- u. Forstw. 5: 489–492. 1907.

[2] Elliott, Charlotte. Manual of bacterial plant pathogens. ix + 349 pp. The Williams & Wilkins Company, Baltimore. 1930. (See page 100).